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DIAGNOSIS OF RUPTURED ABDOMINAL AORTIC ANEURYSM

REPORT OF A CASE

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Rupture of an aneurysm of the abdominal aorta is of infrequent occurrence. The correct diagnosis of this accident has rarely been made. This study is based on a survey of the literature and a personally encountered case. Kampmeier [1] in 1936 reported 73 cases and reviewed the literature on the subject. He found 313 cases reported up to the time of his publication. Since his review, there have been reported 41 additional cases. The total number of cases in the literature is 427. We wish to add 2 cases, 1 of which is reported in detail. The autopsy records of the Mount Sinai Hospital since 1930 have revealed 1 case of rupture of an aneurysm of the abdominal aorta. The second case in the hospital records, described in this paper, makes a total number of 429 reported cases.

Cases of abdominal aneurysm fall into two major groups, those in which the aneurysm is observed before rupture and those in which there are symptoms attributable to rupture of the sac. The violent abdominal symptoms caused by a ruptured aneurysm make it necessary to consider this lesion as one of the possible causes of an acute abdominal catastrophe. The symptoms of rupture are often bizarre and the diagnosis difficult. It is our belief, however, that careful consideration of the clinical findings and proper selection of laboratory and roentgen studies may often lead to the correct diagnosis.

REPORT OF CASE

H. E. C., an obese white man aged 62, was first seen at 7:30 p. m. on June 3, 1938, complaining of excruciating pain of sudden onset in the left side of the abdomen and the left groin. At the onset of the pain he had gone into profound syncope and had a profuse involuntary bowel movement.

From the Mount Sinai Hospital.

1. Kampmeier, R. H.: Aneurysm of Abdominal Aorta: Study of Seventy-Three Cases, Am. J. M. Sc. **192**:97-109, 1936.

The past history revealed the following items of interest: (1) There had been vague digestive complaints for the past two years, usually relieved by sodium bicarbonate; (2) nephropexy on the right side had been performed two years previously; (3) an inguinal hernia on the left side had been repaired twice; (4) a few months previously the patient had been treated for prostatic enlargement, had had an indwelling catheter for ten days and subsequently had had cystitis, and (5) hypertension had been present for a number of years, the systolic blood pressure averaging 160 mm. of mercury.

On the morning of June 3 the patient complained of a dull pain in the left groin, which persisted up to the onset of the sudden, agonizing pain.

When the patient was first seen, he was in extreme shock, pulseless, cold and clammy. The respirations were rapid and shallow, and the blood pressure was unobtainable. The abdomen was soft and not distended. He complained of severe pain when the left lower abdominal quadrant was palpated. A vaguely defined, fixed, nonpulsatile mass could be felt in this region. There was no tenderness over the inguinal scar. No tenderness or rigidity was noted in either costovertebral angle. Peristalsis could be heard over the abdomen. No bruit was present.

The patient was taken to the hospital immediately. He vomited several times en route. On arrival, treatment for the shock was at once instituted, external heat, the Trendelenburg position, stimulants, morphine and intravenous dextrose-saline solution being used. The tentative diagnoses considered were rupture of a peptic ulcer, ureteral stone and mesenteric vascular occlusion. An acute vascular crisis, especially mesenteric vascular occlusion, was considered the most probable diagnosis. A blood count at this time showed: hemoglobin, 86 per cent; red cell count, 4,240,000 per cubic millimeter; white cell count, 24,400 per cubic millimeter, and polymorphonuclear cells, 78 per cent.

Within two hours the patient had responded to treatment. The skin was dry, the pulse was stronger and the blood pressure was 64 systolic and 38 diastolic. A flat plate of the abdomen was taken, and a fluoroscopic examination of the diaphragmatic areas was carried out with the patient in the semiupright position. The report of the roentgenologist follows: "There is no evidence of any gaseous distention of the large or the small bowel. Neither kidney can be distinctly visualized. There is evidence of a faint reniform shadow on the right side, but on the left side there is a suggestion of a mass in the renal region. The right psoas muscle can be faintly outlined. The left cannot be delineated. There is no roentgen evidence of stone in the kidney. Fluoroscopic examination reveals the diaphragm to be normally mobile. There is no evidence of gas under the diaphragm."

On his return from the x-ray room the patient was given an enema, which returned a few small fecal particles but no flatus.

The obliteration of the line representing the left psoas muscle was assumed to be due to a large congested and infarcted area of bowel. The extremely high white cell count was also in favor of the diagnosis of mesenteric vascular occlusion. In view of the patient's extremely critical condition, conservative treatment was given.

The following morning (June 4), his condition remained essentially unchanged. The pulse was still weak and rapid; the temperature was subnormal, and the blood pressure was 50 systolic (diastolic pressure ?). An electrocardiogram taken at this time was reported as falling within normal limits. The sugar content of the blood was 100 mg., the urea nitrogen content 25.5 mg. and the chloride content 595 mg. per hundred cubic centimeters. The Wassermann and Kahn reactions were negative. The patient was placed in an oxygen tent, and treatment with morphine and parenterally administered fluids was continued. At 2

p. m. he voided urine for the first time since admission. One ounce (30 cc.) of cloudy urine was passed, containing a cloud of albumin, many white blood cells and an occasional red blood cell but no sugar or acetone. The possibility of acute pancreatitis was thought of, and the urine was examined for diastase. This substance was observed in dilutions up to 1:50. It was thought that the marked oliguria was probably due to the continued low blood pressure, which resulted in insufficient renal filtration pressure. During this day the patient began to show occasional periods of irrationality. The abdominal findings continued unchanged, although some distention was beginning to appear. The mass in the left lower quadrant persisted. An enema given in the afternoon returned no feces or flatus. A blood count taken during the day showed: hemoglobin 76 per cent; red cell count, 3,810,000 per cubic millimeter; white cell count, 27,500, per cubic millimeter, and polymorphonuclear cells, 80 per cent (30 per cent young forms).

At 9:30 a. m. on the following day (June 5) the patient was catheterized, and the bladder was found empty. The value for urea nitrogen was 37.4 mg. The abdomen showed increased distention, but peristalsis was still audible. A barium sulfate enema showed no abnormalities in the colon. An Abbott tube was introduced nasally, and a large amount of foul-smelling greenish black fluid was evacuated.

In view of the progressively downhill course and the increasing clinical signs of intestinal obstruction, exploratory laparotomy was decided on. Our tentative preoperative diagnosis was mesenteric vascular occlusion.

Operation.—With the region under local anesthesia, a left lower rectus incision was made and the abdomen was explored. The descending colon and the sigmoid were not distended but were pushed forward by an enormous hematoma occupying the retroperitoneal area. Just above the bifurcation of the abdominal aorta a firm, pulsatile mass the size of an orange could be felt. The diagnosis of aneurysm of the abdominal aorta with rupture and retroperitoneal hemorrhage was obvious, and the abdomen was closed without further exploration.

The patient stood the operation well. On his return from the operating room his pulse was 104 and his blood pressure was 95 systolic and 60 diastolic. Later in the evening he was given a slow transfusion of 400 cc. of citrated blood. At 10:45 p. m. his blood pressure was 110 systolic and 85 diastolic. He was catheterized at this time, and 1 ounce (30 cc.) of urine was obtained. At 3 a. m. on June 6 he complained of sudden sharp abdominal pain; the pulse became rapid and feeble, and the blood pressure dropped. It was apparent that further hemorrhage was taking place from the ruptured aneurysm, and therapy was confined to complete morphinization. At 3:25 p. m. the patient died.

Autopsy (Abdomen).—When the abdomen was opened the most striking feature was the bulging anteriorly of the retroperitoneal tissues. The bulging extended upward to within a few centimeters of the diaphragm and laterally to about the midaxillary line. It was more prominent on the left side than on the right. The peritoneum over the bulging area was bluish and tense. On incision the bulging was seen to be due to an extreme infiltration of freshly clotted blood into the retroperitoneal tissues. The hemorrhagic process extended into and involved part of the mesentery of the small intestine. A great deal of clotted blood was observed around the lower pole of the left kidney and the left ureter. There was no free blood in the peritoneal cavity. The hemorrhage was seen to be due to the recent rupture of an aneurysm of the lower portion of the abdominal aorta. The

aneurysm was located about 1.5 cm. above the bifurcation of the aorta. It involved primarily the posterior wall of the aorta and projected posteriorly to the left side. It measured 6 cm. in diameter and was filled with fresh blood clot, which was easily separable from its wall. About 2 cm. below the upper boundary of the aneurysm a partially detached atheromatous plaque was seen. This area communicated directly through the wall of the aneurysm to the densely infiltrated retroperitoneal tissues. The wall of the sac was of about the same thickness as the uninvolved aortic wall, averaging about 3 mm. Both the sac and the aortic

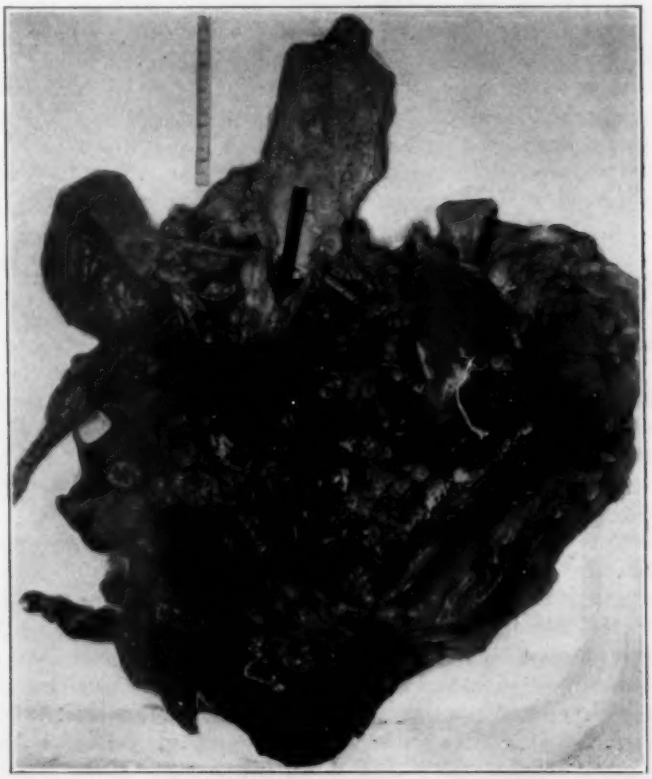

Fig. 1.—Retroperitoneal tissues removed en masse, showing retroperitoneal hemorrhagic infiltration.

wall showed a moderate amount of atheromatous change, but no ulcerations were present except at the point of rupture.

Microscopic Observations.—The pancreas showed fatty infiltration. The kidneys showed arteriolosclerosis, arteriosclerosis and cloudy swelling. There were marked congestion and cloudy swelling of the liver. The spleen showed marked congestion and focal hemorrhage. There was cloudy swelling of the adrenal glands. Medial scarring of the aorta was observed.

DIAGNOSIS

Most ruptured abdominal aortic aneurysms, as in our case, are diagnosed either at the operating table or at autopsy. This lesion has been mistaken for many diseases causing acute abdominal symptoms. Study of the case reports in the literature shows that ruptured abdominal

Fig. 2.—Aorta opened, showing the aneurysmal sac.

aortic aneurysm has been variously diagnosed as ruptured peptic ulcer, ureteral calculus, volvulus of the pelvic colon, acute pancreatitis, mesenteric vascular occlusion, acute intestinal obstruction, perinephritic abscess and psoas abscess.

Ruptured Peptic Ulcer.—A history of gastric complaints is often found in cases of abdominal aneurysm, particularly if the aneurysm is

in the region of the celiac axis. Osler [2] mentioned the fact that gastric symptoms may be early and deceptive. Pressure on nerve plexuses may cause a boring type of pain and spasm similar to those caused by a penetrating ulcer. The initial collapse of ruptured ulcer may simulate the shock of ruptured aneurysm. Rarely, as in the case reported by McLean and Fiddes,[3] the rupture may be intraperitoneal, with all the signs of sudden acute peritonitis. Often it is difficult or impossible to palpate the aneurysm because of variations in its location and size and

Fig. 3.—Aorta opened. Note the probe in an opening in the aneurysm.

because of the fact that expansile pulsation may be either present or absent. Such pulsation is usually not present, since the wall of the sac is frequently partially obliterated by a thick, laminated clot.

2. Osler, W.: The Principles and Practice of Medicine, ed. 12, revised by T. McCrae, New York, D. Appleton-Century Company, 1935.

3. McLean, J. A., and Fiddes, J.: Two Cases of Sudden Death from Rupture of Aorta and from Rupture of an Abdominal Aneurysm, M. J. Australia **1**:807-809, 1929.

Ureteral Calculus.—The radiation of pain in cases of ruptured aneurysm may be similar to that of ureteral colic. Since the rupture is nearly always retroperitoneal, irritation of the spinal nerves and sympathetic plexuses by the retroperitoneal hemorrhagic infiltration may produce pain of variable distribution. In a case reported by Willis [4] the typical symptoms of ureteral calculus were present. At operation a retrorenal hematoma was found, and autopsy disclosed a ruptured saccular aneurysm of the abdominal aorta. The clinical picture of severe shock and hemorrhage should, however, eliminate the diagnosis of ureteral colic in most cases.

Volvulus of the Pelvic Colon.—Intermittent bleeding from a small rupture may simulate the colicky pain of volvulus. In addition, the retroperitoneal hemorrhage may produce an abdominal mass. Cuny [5] reported a case in which, because of colicky pain, an abdominal mass and bloody diarrhea, a diagnosis of volvulus was made. At operation a ruptured aneurysm and a retroperitoneal hematoma were found. Cuny stated that preoperatively an area of ecchymosis was seen in the flank. He expressed the opinion that not enough significance was attached to this observation and emphasized that the sign may have diagnostic importance.

Acute Pancreatitis.—Retroperitoneal hemorrhage from a ruptured aneurysm may produce all the symptoms of acute pancreatitis, especially if the aneurysm or sanguineous infiltration is in the region of the pancreas. In our case, presumably because of irritation of the pancreas by the extravasated blood, the diastase content of the urine was increased. This is the first time this observation has been recorded. Bazy and Calvet [6] reported a case in which laparotomy was performed after a diagnosis of ruptured ulcer had been made. A hemorrhagic infiltration of the pancreas was discovered, and, in addition, an ovoid nonpulsatile mass dorsal to the pancreas was present. The latter was thought to be a pancreatic cyst. Autopsy revealed a saccular aneurysm of the aorta with rupture directly into the substance of the pancreas.

Mesenteric Vascular Occlusion.—The sudden onset of acute abdominal pain, collapse and vomiting and the presence of a high leukocyte count may lead to the diagnosis of mesenteric vascular occlusion. This

4. Willis, P. W.: Ruptured Aneurysm of Abdominal Aorta with Left Retrorenal Hematoma; Symptoms Suggestive of a Right Ureteral Calculus, S. Clin. North America **10**:1231-1234, 1930.

5. Cuny, J.: Rupture d'un anévrysme de l'aorte abdominale simulant un volvulus du côlon pelvien, Lyon chir. **34**:58-59, 1937.

6. Bazy, L., and Calvet, J.: Syndrome abdominal aigu par apoplexie pancréatique (pancréatite aiguë hémorragique) coïncidant avec un anévrysme de l'aorte abdominale, Mém. Acad. de chir. **61**:1336-1340, 1935.

disease occurs in patients of the same age group as most of those with ruptured aneurysm, and patients with either condition usually show evidence of arteriosclerosis and hypertension. Arteriosclerosis is considered by many observers to be the major etiologic factor in abdominal aneurysm, as contrasted with thoracic aneurysm, of which syphilis is the usual cause.[7] The intrasaccular thrombus in aneurysm may obstruct the orifice of the superior or the inferior mesenteric artery and produce the complete syndrome of mesenteric vascular occlusion. In a case reported by Gilmour and McDonald[8] this picture was produced by occlusion of the superior mesenteric artery in the sac of an abdominal aortic aneurysm.

Acute Intestinal Obstruction.—Paralysis of the bowel in cases of ruptured aneurysm may occur as a result of the retroperitoneal hemorrhage, particularly if the leakage is slow and the patient survives for a few days. Vomiting and constipation may be present, as they were in our case. In a case reported by Jaffé[9] a diagnosis of acute intestinal obstruction was made because of vomiting, constipation and signs of peritoneal irritation. Autopsy revealed the usual retroperitoneal hematoma of ruptured aneurysm.

Perinephritic Abscess.—Accumulation of blood in the loin from a ruptured aneurysm may present the picture of a perinephritic abscess. Rusche and Bacon[10] reported a case in which there were pain in the loin, a tender mass in this area, obliteration of the line representing the psoas muscle on roentgen examination, nausea and distention. A lumbar incision disclosed a huge hematoma, and autopsy revealed a ruptured saccular aortic aneurysm. A report by Peel[11] presented a similar case, in which, in addition to the other symptoms mentioned, the typical syndrome of uremia was present.

Psoas Abscess.—A retroperitoneal hemorrhage may burrow along the psoas muscle and present in Scarpa's triangle, simulating a psoas

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7. Neely, J. M.: Ruptured Abdominal Aorta: Clinico-Pathological Study of Five Cases from Lancaster County Medical Museum, Nebraska M. J. **22**:370-377, 1937. Bell, E. T.: A Text-Book of Pathology, ed. 3, Philadelphia, Lea & Febiger, 1938.

8. Gilmour, J., and McDonald, S., Jr.: Aneurysm of Abdominal Aorta and Thrombosis of Superior Mesenteric Artery Associated with Bullet Wound of Lung, Brit. M. J. **2**:587-589, 1932.

9. Jaffé, H.: Rupture of Abdominal Aneurysm Simulating Acute Intestinal Obstruction, Brit. M. J. **1**:1173, 1925.

10. Rusche, C. F., and Bacon, S. K.: Ruptured Abdominal Aortic Aneurysm Simulating Perinephritic Abscess, with Report of a Case, Brit. J. Urol. **7**:330-332, 1935.

11. Peel, J. H.: Rupture of Aneurysm of Abdominal Aorta, Lancet **1**:512, 1932.

abscess. Eckert and Baker [12] reported a case in which the diagnosis of psoas abscess was made because of abdominal, lumbar and femoral pain, nausea, vomiting and a pulsatile mass in the femoral region. Lumbar incision revealed a massive hematoma from a ruptured aneurysm of the abdominal aorta.

COMMENT

Although we are concerned in this report with a study of ruptured abdominal aneurysm, it is interesting, in order to illustrate the difficulty of the diagnosis, to note a few of the conditions with which unruptured aneurysm of the abdominal aorta has been confused. This condition has been variously diagnosed as tumor of the small bowel,[13] tumor of the spinal cord,[14] spinal arthritis, tumor of the liver, carcinoma of the stomach, pancreatic cyst, malignant tumor of the retroperitoneal nodes, renal tumor [1] and, in fact, almost every intra-abdominal and retroperitoneal syndrome known.

A clinical analysis of the cases reported in the literature and of our personal case has led us to the opinion that certain features of this condition are sufficiently distinctive to bring the condition to mind as a possibility in the diagnosis of obscure acute abdominal syndromes. Two general features are important: (*a*) the fact that ruptured aortic aneurysm is an acute vascular disease and presents features that characterize vascular crises in general and (*b*) the fact that the retroperitoneal hemorrhage usually present causes certain signs and symptoms that differentiate it from intraperitoneal disease.

The pain of all vascular crises is sudden and violent. The great majority of ruptured aneurysms evidence themselves first with sudden, agonizing pain, usually abdominal but occasionally lumbar as well. Accompanying the pain is shock, usually severe and persistent. The clinical signs of intraperitoneal disease are slight or absent, the lack of tenderness or rigidity being in great contrast to the severity of the abdominal symptoms. A localized, fixed mass is often present. If this shows expansile pulsation and a bruit, the diagnosis is obvious. Unfortunately, in many cases neither of these signs is present. Nausea, vomiting and distention due to irritation of the retroperitoneal nerve plexuses are common but not marked. Intestinal peristalsis may be but little affected.

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12. Eckert, G. A., and Baker, R. E.: Rupture of Aneurysm of Abdominal Aorta from Surgical Viewpoint: Report of Two Cases, U. S. Nav. M. Bull. **29**: 667-671, 1931.

13. Petridis, P.: Sur cinq cas d'anévrysme rompu de l'aorte dont quatre de l'aorte abdominale et un de l'aorte thoracique, J. Egyptian M. A. **13**:44-64, 1930.

14. Weingrow, S. M., and Bray, W. A.: Aneurysm of Abdominal Aorta: Case Report, Am. J. Roentgenol. **36**:194-196, 1936.

The leukocyte count is usually high, a common finding in cases of internal hemorrhage. This observation has been noted by many observers and was a feature of our case.

The roentgen studies are most significant. Perforation of a hollow viscus can be ruled out in the majority of cases by the absence of free gas under the diaphragm. A flat plate of the abdomen often shows obliteration of the line representing the psoas muscle.[15] This observation directs attention toward the retroperitoneal area. Volvulus and obstruction of the large bowel can be immediately dismissed if the colon is roentgenographically normal after a barium sulfate enema. The most important roentgen study in cases of suspected aneurysm is that which gives a lateral view of the lower thoracic and the lumbar vertebrae. Erosion of the vertebral bodies with preservation of the intervertebral disks[16] in the presence of suggestive symptoms is almost pathognomonic of aneurysm.

The finding of a moderately increased diastase content of the urine, as far as we have been able to determine, has not been previously recorded. Since the pancreas is entirely retroperitoneal and the hemorrhage present in ruptured aneurysm is similarly retroperitoneal, pancreatic irritation, as evidenced by increased diastase in the urine, should be helpful in the diagnosis of this lesion.

Treatment.—The treatment of abdominal aneurysm has given most disappointing results. For many years attempts have been made to attack this lesion by ligation, both proximal and distal, by proximal compression with aluminum and fascial bands, by the Moore-Corradi method of wiring and electrolysis and by the introduction of Colt's cages. . Reid[17] reported 4 cases of abdominal aortic aneurysm in which ligation with tapes and metallic bands was done. All the patients died, 3 from secondary hemorrhage caused by cutting through of the aorta by the band. The same author reported 8 cases of abdominal aneurysm wired by the Moore-Corradi method, with no cures. Colt[18] introduced a wire cage into the aneurysm through a specially designed stilet in 2 cases; both the patients died. Power[19] had more success with this

15. Held, I. W., and Goldbloom, A. A.: Three Rare Intra-Abdominal Cases, S. Clin. North America **14**:389-405, 1934. Rusche and Bacon.[10]

16. Brailsford, J. F.: Aneurysm of Abdominal Aorta: Diagnosis by Lateral Radiograph of Spine, Brit. J. Surg. **14**:369-371, 1926. Weingrow and Bray.[14]

17. Reid, M. R.: Aneurysms in the Johns Hopkins Hospital: All Cases Treated in the Surgical Service from the Opening of the Hospital to January 1922, Arch. Surg. **12**:1-74 (Jan., pt. 1) 1926.

18. Colt, G. H.: Aneurysm of Abdominal Aorta, Brit. J. Surg. **13**:109-113, 1925.

19. Power, D'A.: The Palliative Treatment of Aneurysm by "Wiring" with Colt's Apparatus, Brit. J. Surg. **9**:27-36, 1921.

method, having 4 survivals in 11 cases. Brooks [20] treated an aneurysm successfully by proximal ligation, using a broad fascial strip. Two other successful ligations have been recorded,[20] 1 by Matas and 1 by Vaughan.

This brief review of the results of surgical treatment of unruptured aneurysm of the abdominal aorta indicates that once this lesion has ruptured the condition becomes practically hopeless. In only 1 case reviewed by us has direct attack been made on a ruptured aneurysm. This was a case reported by Petridis; [13] death of the patient followed immediately. It is within the realm of possibility that absolute rest may suffice to seal the perforation in the aneurysmal sac and may result in recovery. A case reported by Leriche [21] in which a ruptured aneurysm was found to have sealed itself off by the formation of a second, false aneurysm which did not rupture until operative intervention was attempted illustrates the rationale of judicious neglect. Certainly the results of nonoperative treatment can be no worse than those of operation.

CONCLUSIONS

1. Ruptured abdominal aortic aneurysm should be considered in the diagnosis of any puzzling acute abdominal crisis.

2. The distinctive features are those of vascular crisis, shock and retroperitoneal hemorrhage.

3. Obliteration of the line representing the psoas muscle and erosion of the vertebral bodies with preservation of the intervertebral disks are important roentgen observations.

4. A high leukocyte count is constant.

5. The diastase content of the urine may be moderately elevated.

6. The treatment advised is nonoperative.

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20. Brooks, B.: Ligation of the Aorta: A Clinical and Experimental Study, J. A. M. A. **87**:722-725 (Sept. 4) 1926.

21. Leriche, R.: Opérations pour rupture d'anévrisme de l'aorte, avec formation d'hématome enkysté à évolution lente (deux observations), Bull. et mém. Soc. nat. de chir. **60**:876-878, 1934.

TRAUMATIC FAT EMBOLISM

REPORT OF TWO CASES WITH RECOVERY

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Fat embolism is a definite, well established disease entity which is neither new nor infrequent.[1] Over seventy-five years ago the condition was reported in man; its clinical aspects were noted, its physiologic alterations described and the postmortem observations recorded. Many articles have appeared in the literature since that time covering the various aspects of the condition. From them one is able to draw a fairly definite and accurate picture of what happens in a person in whom this complication develops. The term complication is used because in nearly all cases it is a complication of some other condition in the body, the exception to this being fat embolism due to intramuscular injections of medicated oils that inadvertently enter the blood stream. The conditions to which it is a complication are diversified and include osteomyelitis, nephritis, burns, orthopedic operations, operations on and injuries to fatty tissues, fractures, contusions and degenerative processes in the body. Interest here is in traumatic fat embolism, especially that due to a fracture.

That the introduction of liquid fat into the blood stream follows injuries to the skeletal system with surprising frequency has been definitely proved at autopsy. Not all fat embolisms, however, are of such severity as to cause death or even to produce clinical symptoms. Many persons who live have few or no symptoms, and many others who have symptoms recover without the exact nature of the condition being diagnosed.

Read before the Harlem Surgical Society, Oct. 19, 1938.

From the Surgical Service of the Harlem Hospital, Dr. Louis T. Wright, Director.

1. (*a*) Gauss, H.: The Pathology of Fat Embolism, Arch. Surg. **9**:593 (Nov., pt. 1) 1924; (*b*) Studies in Cerebral Fat Embolism, with Reference to the Pathology of Delirium and Coma, Arch. Int. Med. **18**:76 (July) 1916. (*c*) Groskloss, H. H.: Fat Embolism, Yale J. Biol. & Med. **8**:59, 175 and 279, 1935. (*d*) McCaster, J. C.: Fat Embolism: A Case Report of Cerebral Involvement, Wisconsin M. J. **36**:724, 1937. (*e*) Warthin, A. S.: Traumatic Lipaemia and Fatty Embolism, Internat. Clin. **4**:171, 1913. (*f*) Watson, A. J.: Fat Embolism: Report of a Case with Review of the Literature, Brit. J. Surg. **24**:676, 1936. (*g*) Wright, R. B.: Fat Embolism, Ann. Surg. **96**:75, 1932. (*h*) Vance, B. M.: Significance of Fat Embolism, Arch. Surg. **23**:426 (Sept.) 1931.

There has been dispute as to the origin of the fat, it being claimed by some that there is not enough fat in any long bone of the body to cause death even if all of it should enter the circulation. Whether the injured bone is the sole source of the fat or whether there is some additional alteration in the normal lipoid content of the blood, the chief source of the fat is the injured bone. In that area the fat globules are liberated and are either drawn or forced by compression into the torn haversian veins. A small amount may reach the circulation by way of the lymphatics. The fat passes to the right side of the heart and from there enters the pulmonary circulation, where it becomes lodged in the finer arterioles and capillaries. If the amount of fat is too great, it may act much the same as an air embolus on the heart and cause death before it reaches the lung.

As the pressure in the pulmonary circulation rises, many of these fat globules are forced on through the capillaries of the lungs and are carried to the left side of the heart, from whence they may go to any part of the body but chiefly to the brain and kidneys.

To understand the symptoms produced by fat embolism, it is necessary first to understand the pathologic process. The entire clinical picture is the sum total of the effects of numerous small, transient emboli lodged in capillaries, each of which has practically the same unit pattern, the symptoms produced being dependent on the number, location and duration of the emboli. This unit pattern consists essentially of a small vessel obstructed by a fat globule and surrounded by extravasated blood, causing focal anemia, edema or necrosis. Later various white blood cells and phagocytes appear in the area to help in repair.

Shortly after the fat enters the systemic circulation it begins to be excreted by the kidneys and can be detected in the urine. This process of elimination is aided by the absorptive and phagocytic actions of various liver cells and by phagocytes and giant cells which invade the damaged areas. Enzymes in the blood stream aid the process by saponification of the fat.

Although fat globules reach practically all parts of the body, symptoms are usually referable to the lungs and brain. Consequently, there are two types of fat embolism clinically, the pulmonary and the cerebral, depending on the preponderance of one group of symptoms over the other. However, no sharp line can be drawn between the two, as there are some pulmonary and some cerebral symptoms in each case.

Symptoms develop within a few hours to a few days. There is always a free interval, a kind of incubation period, which is an aid in making a differential diagnosis. With the pulmonary type of embolism, respiratory and cardiac embarrassment are evident. There are dyspnea, cough, cyanosis, restlessness and a feeling of constriction in the chest. Air hunger may develop. The pulse is rapid and may be irregular. The temperature may be normal, but in the great majority of cases it is

elevated. The sputum becomes frothy and may be blood streaked. Stained with scarlet red or sudan III, it may show fat globules. Rales are heard scattered over the pulmonary fields as a result of mild or severe pulmonary edema. The right side of the heart is put under considerable strain and is usually dilated. Blood pressure is most frequently low.

With the cerebral type, drowsiness, disorientation, stupor and coma appear, in addition to some respiratory symptoms. Hallucinations or delirium may be evident. There are no persistent localizing neurologic symptoms, but transient muscle spasm, tremors, convulsions and paralyses may occur. All are indicative of widespread cerebral involvement and irritation. Incontinence of feces and urine often develops. In most cases of the cerebral type and in some of the pulmonary type of embolism petechial hemorrhages of the skin and conjunctivas develop. This is a sign of great diagnostic importance.

As previously stated, nearly all persons with fat embolism recover without a diagnosis being made, or a diagnosis is made after the postmortem examination. Consequently it is not possible even to approximate the true mortality rate. On the basis of the cases reported in the literature, this has been placed at from 85 to 90 per cent. There are relatively few cases in which recovery was reported; only 1 such case could be found in the American literature. Two patients with this condition were seen in the wards of the Harlem Hospital during the past two years, both of whom recovered. These 2 cases are here reported.

REPORT OF CASES

CASE 1.—T. B., a 22 year old Negro student, sustained a fracture of both bones on the right leg in an intercollegiate basketball game near midnight, Feb. 13, 1937. He was brought to the Harlem Hospital immediately with his leg in an improvised splint. His general condition was good. There were no signs of injury aside from swelling, tenderness and ecchymosis of the right leg. Roentgen examination confirmed the diagnosis of fracture of the tibia and fibula. The temperature was 98 F., the pulse rate 68, the respiratory rate 18 and the blood pressure 132 systolic and 84 diastolic. The urine and the blood count were normal. A circular cast was applied from the middle of the thigh to the toes without anesthesia. The cast was split anteriorly in its entire length.

The next morning the patient's condition was the same, but by afternoon the temperature had risen to 102.4 F. The pulse rate was 88, and the respiratory rate 22. The white cell count was 17,600, with 84 per cent polymorphonuclears. The patient was somewhat drowsy.

On the evening of the following day, two days after admission, drowsiness was marked, and dyspnea and cyanosis were present. The temperature was 102 F., the pulse rate 92 and the respiratory rate 40. There were numerous moist and coarse crepitant rales heard throughout both pulmonary fields. A tentative diagnosis of fat embolism was made.

The next morning the temperature was 101 F. and the respiratory rate 36. Rales were present but were fewer. Drowsiness had increased, but the patient could be awakened. A physician from the pneumonia service who was called in said that

pneumonia was not present. Culture of the sputum in mouse peritoneum was negative for pneumococci. Oxygen was given by nasal catheter. A roentgenogram of the chest, made with the portable apparatus, showed a few patches of consolidation scattered over both pulmonary fields and a general haziness that is often seen in cases of pulmonary edema. Two petechiae were noted on the conjunctiva of the right lower lid. By evening they had become much more numerous. None was found on the skin. There was some expectoration of bloody sputum. Cultures of the blood were taken and later reported to be negative.

On the fourth day after injury the temperature was almost normal, the dyspnea had disappeared, and all signs in the chest were gone. The subconjunctival hemorrhages continued for several days longer. All examinations of the urine were negative for fat.

Considering the trauma, the free interval, the onset of drowsiness, the dyspnea and cyanosis, the rales indicative of pulmonary edema, the

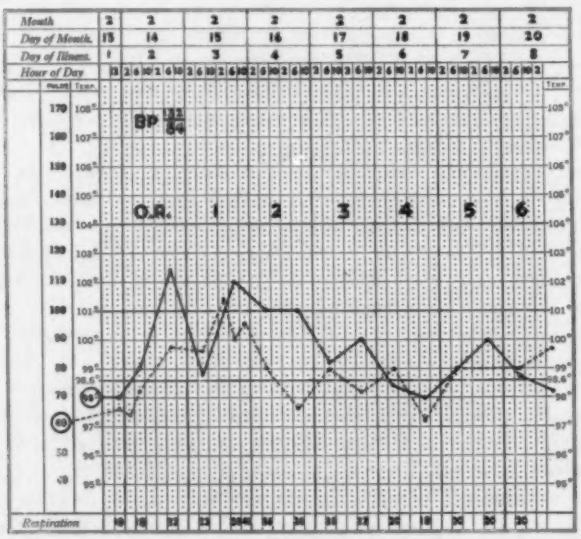

Fig. 1 (case 1).—Temperature chart.

petechial hemorrhages of the conjunctiva, the brevity of the condition and the absence of any other disease that would produce these symptoms, the diagnosis of fat embolism of the pulmonary type was justified.

CASE 2.—B. A., a 19 year old Negro youth, was admitted to the surgical service of the Harlem Hospital, at 2:30 a. m. on June 27, 1938, one hour after being hit by an automobile. He had been transported a distance of about half a mile with his legs in Thomas splints. There was no history of bleeding or unconsiousness.

Physical examination revealed a well developed youth in good physical condition, except for the pain in his legs. There were some abrasions and contusions of the supraorbital and infraorbital regions. The left wrist showed a small laceration. There were abrasions on both knees. Each leg showed deformity and tenderness at the junction of the middle and the lower third. A diagnosis of fracture of both legs and multiple contusions and abrasions was made. The temperature on admission was 97 F., the pulse rate 88, the respiratory rate 22 and the blood

pressure 150 systolic and 100 diastolic. The white cell count was 6,700, with 68 per cent polymorphonuclears, 30 per cent lymphocytes and 2 per cent transitionals. The red cell count was 4,700,000, with a hemoglobin content of 70 per cent. The Kahn reaction of the blood was negative on two occasions. Roentgenograms of the legs showed a fracture of each tibia at the junction of the middle and the lower third, in good position.

The patient was taken to the operating room at 4 a. m., and a circular plaster of paris cast was applied to each leg from the middle of the thigh to the toes, with the knee slightly flexed and the ankle at right angles. The casts were split anteriorly in their entire length. No anesthesia was used. The patient was returned to the ward at 5:30 a. m. in good condition.

At 11 a. m., about ten hours after the injury, the patient seemed to be in a daze. He complained of pain in the back and abdomen. He had been unable to void urine since admission. At 5 p. m. he was extremely restless. The bladder was catheterized, and 6 ounces (177 cc.) of amber-colored fluid was obtained. The urine was normal. At 7 p. m. the patient calmed down and was sleeping. At 8:30 p. m. he seemed irrational and was talking at random and complained of pain in the back and both loins. The temperature was 102.8 F., the pulse rate 110. the respiratory rate 24 and the white cell count 16,400, with 88 per cent polymorphonuclears. Roentgenograms of the spine and pelvis were normal. Codeine and phenobarbital were given.

The patient slept fairly well, but the next morning, June 28, he was still disorientated, and by 9 a. m. he was in a deep stupor and could not be aroused. There were spasmodic contractions of the upper extremities and occasional slight tremors of the body. There was incontinence of urine. A spinal tap yielded clear fluid under a slight increase of pressure. The abdominal wall was rigid, retracted and markedly tender. The temperature at this time was 101.6 F., the pulse rate 102 and the respiratory rate 22. There were numerous petechial hemorrhages over the upper part of the chest. One such hemorrhage was seen on the conjunctiva of the left eye. There were no other neurologic symptoms. A diagnosis of fat embolism of the cerebral type was made. Examination of the urine gave negative results. The blood showed 174 mg. of cholesterol and 197 mg. of fatty acids per hundred cubic centimeters. A continuous infusion of 5 per cent dextrose in physiologic solution of sodium chloride was started, and alcohol sponges were ordered for the control of high temperatures. The patient remained in this comatose condition throughout the day and night. The temperature rose to 104.4 F. in the afternoon and fell gradually to 101.8 F. by the next morning. The respiratory rate was 36 in the afternoon, 40 in the evening and 30 the next morning. The patient continued to have urinary incontinence. Roentgenograms of the chest taken at this time were not satisfactory but were sufficiently clear to eliminate the presence of pneumonia. Physical signs of pneumonia were absent.

On the morning of June 29, two days after admission, the patient was still comatose. The temperature was 101.8 F., the pulse rate 106 and the respiratory rate 30. The petechial hemorrhages had become much more numerous on the upper part of the chest and had begun to appear on the neck and lower part of the face and abdomen. A biopsy was taken from the chest in an area of most numerous petechiae.

Later in the morning the patient was more reactive to painful stimuli. In the afternoon he seemed to come out of the coma and at times moved his lips as if attempting to talk, but no sound was audible. By evening, however, he had again lapsed into coma, and there was fecal as well as urinary incontinence.

The following morning, June 30, the temperature had fallen to 101 F., but rose in the evening to 102.4 F. Chemical examination of the blood showed 1.3 mg. of

creatinine, 15 mg. of urea nitrogen and 100 mg. of sugar per hundred cubic centimeters of blood.

On the morning of July 1 the patient's condition was precarious. The temperature was 104 F., the pulse rate 160 and the respiratory rate 36. The petechial hemorrhages had increased in number and were present on the arms and forearms. The pupils were in middilatation, equal and sluggish in response to light. There was a soft systolic murmur at the apex of the heart. Respirations were shallow, and breath sounds were suppressed throughout the chest. Cultures of the blood and of the spinal fluid were made, the spinal fluid was examined and a Felix-Weil test was done. The results of all were later reported to be negative. By evening the temperature was 105.4 F., the pulse rate 130 and the respiratory rate 30.

Throughout the next day, July 2, the temperature remained around 105 F. and the respiratory rate from 32 to 40. Urinary and fecal incontinence continued.

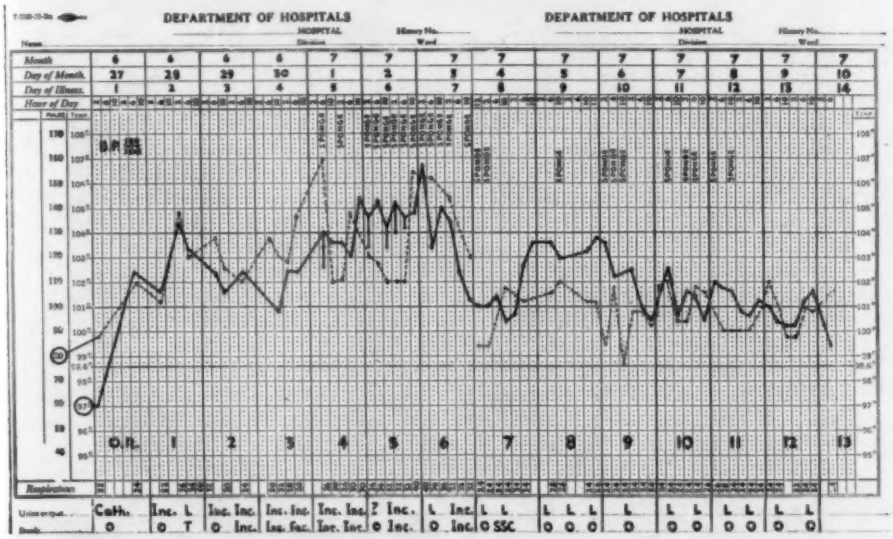
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Fig. 2 (case 2).—Temperature chart. *Inc.* stands for incontinence; *L,* for lost; *SSC,* for soapsuds clysis; *Cath.,* for catheterized; *O,* for none.

On the morning of July 3, at 2 a. m., the temperature was 106.8 F., the pulse rate 154 and the respiratory rate 40. At 6 a. m. the temperature had fallen to 103.4 F., and at 6 p. m. to 101 F. Urinary and fecal incontinence were still present. In the evening the patient rallied, came out of the coma and attempted to speak. He drank 50 cc. of water, the first he had taken by mouth since the day of admission.

On July 4 his condition was improved, although the temperature rose in the evening to 103.6 F. On this day the patient regained control of the bladder and rectal sphincters after five days of incontinence. The rigidity in the arms was entirely absent, but there was extreme weakness in the muscles.

On July 5 he was still conscious and more alert and responded to questions with a nod of his head. He was able to drink water in small amounts. The temperature was 103.6 F., the pulse rate 106 and the respiratory rate 32.

On July 6 the temperature was 103.6 F. The patient was alert but throughout the morning failed to answer questions. For the first time the urine gave a positive reaction for fat.

On July 7 there was considerable improvement. The temperature ranged between 102.6 and 104 F., and the respiratory rate was 24.

On July 8 the condition was improved, and the patient was more alert. The hemorrhagic areas remained only on the shoulders and neck, but instead of areas of discoloration there were present brown, flat-topped, firm papules about a millimeter in diameter.

Gradual improvement continued through the next two days, although the temperature remained at 101.4 F. On the morning of July 10 the temperature was 99.4 F., the pulse rate 106 and the respiratory rate 24. On July 11 the temperature was 99.2 F. The patient began to mumble answers to questions and took fluids by mouth frequently. Intravenous infusions were stopped.

Fig. 3 (case 2).—Petechial rash.

During the next week the temperature ranged between 99 and 100 F., and the general condition gradually improved. The patient began to regain the use of his arm, and the hemorrhagic areas were faint. During the nights and occasionally in the days the patient showed periods of boisterousness and anger. Otherwise convalescence continued to be uneventful.

There can be no doubt but that this case is one of traumatic fat embolism of the cerebral type. The symptoms are exactly similar to those described in the cases in which the diagnosis was proved at autopsy. The extensive petechial hemorrhages and the fat in the urine make the diagnosis certain.

The treatment for fat embolism has been concisely expressed by Vance,[1h] who stated: "As a rule, when the fat has entered the blood stream, the result must be left to the natural defenses of the body, and

only symptomatic treatment can be applied." This was true in the present case. A continuous infusion of 5 per cent dextrose in physiologic solution of sodium chloride was given for two full weeks, the amount averaging about 4 liters a day. Alcohol sponges at intervals of four hours were given to combat high temperatures. Excellent nursing care was administered day and night, and this aided more than any other therapeutic measure in the recovery.

It is interesting to speculate as to the part played by the casts in the duration and severity of the illness, and this patient had on two casts. The veins of the haversian canals do not collapse as veins elsewhere when torn across but remain open and render access to the circulation easy for the fat globules. The extravasation of blood and serum into the tissues exerts a pressure on this liberated fat, tending to force it into the veins. If a cast is applied before the tissues about the injured bone have reached their maximum swelling, the continued extravasation into the tissues causes the pressure to be increased to a tremendous extent and can well be a factor in increasing and prolonging if not in actually causing the introduction of fat into the circulation. More can be done in preventing than in treating fat embolism; and one of the most important points is the careful handling and manipulation of injured bone, as has been pointed out by all writers on the subject. To this can be added the avoidance of any constricting appliance that will increase the intraosseous pressure until after the maximum swelling has been reached. After the diagnosis had been made in this case, the casts should have been removed and the legs left in basket splints until the symptoms of embolism had cleared.

The papular change in the petechiae has not been described before. A biopsy of the skin was made, but nothing of significance was found. It seems possible, however, that these papules represented foreign body reactions to the fat emboli lodged in the vessels of the skin.

Another interesting development in this case was the onset of severe pain in the loins and abdomen twenty-four hours after admission. The abdominal pain and rigidity were so severe that had they been present on admission an exploratory laparotomy would most surely have been done. The pain in the loins was associated with the fact that on catheterization eighteen hours after injury only 6 ounces of urine was obtained, showing a suppression. These symptoms pointed to severe involvement of the renal and mesenteric vessels.

COMMENT

It is hoped that these 2 cases will help to dispel the idea of the rarity and hopelessness of fat embolism and cause those entrusted with the treatment of the injured to bear the condition in mind. Warthin [16] called it a neglected branch of surgery. There is no reason for its continuance as such.

ANEURYSM OF THE SPLENIC ARTERY

REPORT OF A CASE AND REVIEW OF THE LITERATURE

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AND
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BUFFALO

Aneurysm of the splenic artery is a rare disease. It is so dangerous that only early diagnosis and proper treatment can prevent a tragic end. It is seldom diagnosed when encountered clinically, because it is not considered in the differential diagnosis. The entity, because of its rarity, is seldom discussed in the current literature and in many instances is not mentioned in textbooks. It is for these reasons that the following report is presented.

REPORT OF CASE

A white woman aged 30 was first seen about 9 p. m. on January 14, complaining of mild pain in the upper part of the abdomen. She had been well until one week prior to examination (approximately January 7), when she noticed roughness of the throat and "sore glands in the neck." This condition subsided within a few days. She continued to be fairly well until January 13, when she "just didn't feel right." She had the same peculiar feeling on the morning of January 14, and during the latter part of the afternoon she noticed mild aching pain in the upper part of the abdomen. The pain did not radiate. Urgency and frequency of urination were observed. The patient had her supper and about two hours later vomited. There was no fresh blood or coffee ground material in the vomitus. The abdomen became slightly distended and with an enema considerable flatus and dark brown formed stool were passed, with some relief. The abdominal pain and the urgency and frequency of urination persisted.

The systemic review gave negative results except for the following observations: There was moderate dyspnea on exertion. The stools had been dark since the patient had begun taking medicine for "anemia." Since the onset of the present illness urgency and frequency of urination had been present, but there was no burning. Only a few drops to a small amount of urine was passed each time. There was no difficulty in starting the stream. The menstrual periods occurred regularly every twenty-eight days. The last period had started December 20.

The patient had had rheumatic fever when a child and since then had had a "bad heart" and had always been sickly. An appendectomy had been performed when she was 12 years of age, and a cesarean section and ligation of tubes had been done when she was 28, because of the cardiac condition. She had had pyelitis six months prior to examination.

The temperature was 98.4 F., and the pulse rate was 70. The patient was well nourished and fairly well developed. She was in bed but was neither acutely

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ill nor in distress. The skin, conjunctivas and nail beds were normally pink. (These were examined in particular because of the aforementioned anemia, the dark stools and the medicament which the patient was taking. The tablets were of the shape and color of some proprietary tablets containing ferrous sulfate.) The remainder of the physical examination gave negative results except for the following observations: The heart was enlarged to the left; the rate was 70, and the rhythm was regular. There was a rough apical murmur, which was not transmitted. The pulse was regular and of good volume. The abdomen was rounded but not distended. There were a scar over McBurney's point and one scar low in the midline, both of which were pink and firm. No hernias could be detected. The abdomen was soft throughout. In the righ upper quadrant and toward the midline, tenderness was elicited on deep pressure. No masses could be palpated. Slight tenderness was elicited in the right costovertebral angle. No tenderness was present in the left angle.

The impression at this time was as follows: In view of the history of pyelitis six months previously, roughness of the throat and cervical adenitis one week previously, abdominal pain, urgency and frequency of urination and tenderness of the right upper quadrant of the abdomen and the right costovertebral angle at the time of examination, it was felt that a recurrence of the renal infection was probably developing. In spite of the urgency and frequency of urination, a sample of the urine could not be obtained.

The patient was seen again at approximately 2 a. m. on January 15. The abdominal pain became more severe and shot across the upper part of the abdomen. At times the patient complained that it "cut off her breath." The urgency and frequency of urination persisted. The patient stated that there were no other symptoms. The temperature, pulse rate and respiratory rate were normal. The remainder of the physical examination gave results identical with those of the previous examination except that the tenderness in the upper middle part of the abdomen and in the right costovertebral angle was more marked.

About 7 a. m. on January 15, the patient was complaining of severe pain in the lower part of the abdomen which spread along the left side of the abdomen. The pain was much more severe than that complained of earlier in the evening. This sharp, severe pain in the lower part of the abdomen occurred suddenly about 6:30 a. m. and was followed by vomiting, the emesis containing neither old nor fresh blood. After the onset of the pain, the patient's family noticed a gradual change in her appearance. She was extremely pale, all color having disappeared from the skin, nail beds and conjunctivas. She was covered with cold perspiration. The pulse was rapid, and the volume was considerably less than on previous examinations. The abdomen was flat, and in both lower quadrants there was exquisite tenderness to moderate pressure. There was involuntary spasm but no rigidity. Examination of the pelvis revealed no fresh bleeding and no masses, but there was marked tenderness in both fornices, especially the left, and on motion of the cervix.

The patient was immediately taken to the hospital. On her arrival the pulse rate was 140 and the pulse was thready. The respiratory rate was 30. The blood pressure was 78 systolic and 58 diastolic. The color, the cold, clammy skin and the abdominal signs were the same as before. The red blood cell count was 2,280,000 per cubic millimeter; the hemoglobin content was 48 per cent, and the white cell count was 7,300. Intravenous administration of dextrose and saline solution was started immediately. Soon afterward the pulse rate dropped to 92, the volume improved and the blood pressure increased to 88 systolic and 68 diastolic. Suddenly the pulse again became rapid and thready. The patient sank rapidly and died before a transfusion could be given.

The clinical impression on the patient's admission to the hospital was, in addition to rheumatic endocarditis, "ruptured ectopic pregnancy." However, the subsequent course was much too rapid and severe for the latter, and it was felt that the patient had an exsanguinating intraperitoneal hemorrhage, the source of which was unknown.

Autopsy.—General Observation: The skin was extremely pale. Little blood was observed in the vessels.

Heart: The heart weighed 310 Gm. The pericardial fat was preserved. The endocardium of the right atrium showed fatty patches. The tricuspid valve was thin and delicate. The wall of the right ventricle showed fatty infiltration. The mitral valve was thickened on its free margin. Along the line of closure of the anterior leaflet, especially where it joined the posterior leaflet, and along the line of closure of the posterior leaflet there were pinpoint-sized to pinhead-sized glistening gray vegetations. The larger of these were slightly polypous. The surfaces of a few were red. Recent hemorrhages were noted in the endocardium of the left ventricle. The myocardium was pale and showed patchy scarring. The aortic cusps showed small conglomerate vegetations in the noduli Arantii. The aorta measured 6 cm. above the valve. There was slight atheromatosis just above the sinuses, with pinhead-sized patches.

Lungs: There were delicate fibrous bands running from the anterior surface of the upper lobe of the right lung to the parietal pleura. A few fibrous bands were present between the upper and the lower lobe. The interlobular fissure showed petechial hemorrhages. The mediastinal surface of the right lung was adherent to the mediastinum.

Peritoneal Cavity: The peritoneum was bluish. When the cavity was opened, a large amount of fluid and clotted blood was found in the pelvis and in both subphrenic spaces. More than 2 quarts of blood was removed from the general peritoneal cavity. In the gastrohepatic ligament, along the lesser curvature of the stomach as far as the pylorus, but especially around and to the left of the celiac plexus, there was hemorrhage. The lesser sac contained free and clotted blood. Hemorrhage extended through the hiatus of the diaphragm surrounding the esophagus.

Splenic Artery: The artery passed downward and slightly to the left for a distance of 2 cm., where it turned at almost right angles to the left. At this right angle turn opposite the origin of the splenic artery there was an aneurysm which could be measured only with difficulty. It was approximately 2.5 cm. in circumference. It was filled with laminated, somewhat soft mixed clot. This clot was adherent to the inner surface. The wall could not be followed with certainty around the whole aneurysm because of a rupture in the superior posterior part and also in the inferior part. The ruptured aneurysm pressed into the pancreas about 8 cm. from the tail. The pancreas at this point was atrophic. The horizontal course of the splenic artery was normal.

Spleen: The capsule was wrinkled, and there was marked anemia. The organ measured 15 by 6 by 3.3 cm. The splenic vein was patent.

Stomach: The stomach was contracted and compressed along the lesser curvature by the hemorrhage (fig. 2). The serosa was infiltrated with blood.

About 9 cm. from the cardiac orifice there was an indentation into the stomach at its lesser curvature and posterior wall measuring 3.3 by 2 by 1.5 cm., brought about by the aneurysm. The stomach was pushed down by the hemorrhage.

Abdominal Aorta: The aorta showed slight atheromatosis.

Kidneys: Each kidney measured 11.5 by 3.5 by 2.8 cm. The capsules were thin and stripped easily. Embryonal lobulations persisted. Both kidneys were

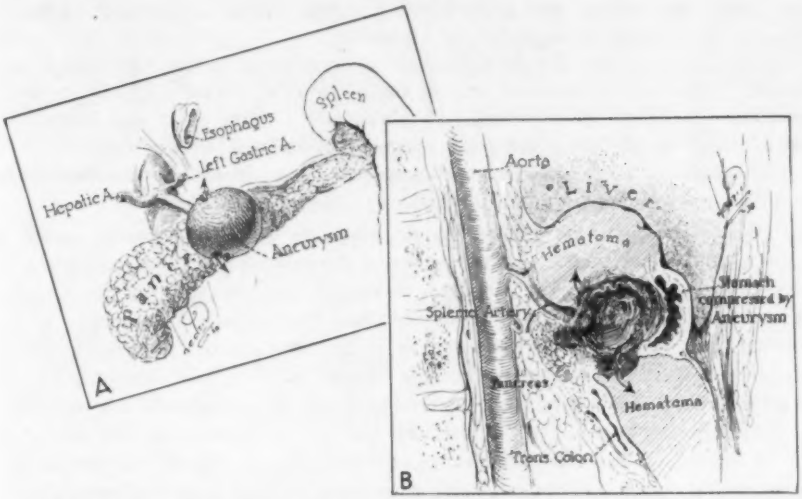

Fig. 1.—*A*, sketch showing the relative position of the aneurysm with the two sites of rupture. *B*, sketch of the lateral view, showing the aneurysm, compression of the stomach, superior and inferior ruptures and subsequent hematomas.

Fig. 2.—Liver lifted upward. Note the dark, glistening hematoma in the gastrohepatic ligament pushing the stomach downward.

very pale. The pelves and ureters were normal. The left kidney showed a depressed scar which measured 1 by 0.5 cm.

Uterus: There were fibrous adhesions between the uterus and the pelvic peritoneum. There was a depressed scar in the midanterior surface, 2.5 cm. in length and 1 cm. from the superior margin. The continuity of the tubes was lost for the first 3.5 cm., but the preserved parts measured 8 cm. and were normal.

The remainder of the postmortem examination gave essentially negative results except for marked anemia of all the viscera.

Pathologic Diagnosis.—The pathologic diagnosis was (1) ruptured aneurysm of the splenic artery 2 cm. from the origin of the celiac axis, filled with laminated clot; (2) protrusion of the ruptured aneurysm into the wall of the stomach and into the pancreas, causing indentation into the stomach and atrophy of the pancreas; (3) hemorrhage into the serosa of the stomach and pancreas about the aneurysm; recent hemorrhage into the lesser sac; distinct hemorrhage into the gastrohepatic ligament; extension through the diaphragm about the lower end of the esophagus; (4) massive hemorrhage into the peritoneal cavity, with more than 2 quarts of fluid and clotted blood; (5) chronic rheumatic endocarditis of the mitral and aortic valves, with a number of firm gray vegetations; (6) petechial hemorrhages in the endocardium of the left ventricle; small patchy scarring of the myocardium in the posterior wall of the left ventricle; (7) fibrous bandlike pleurisy of the right lung to the parietal pleura and between the lobes; petechial hemorrhages in the right pleura; (8) depressed scar of the left kidney, probably following infarction; (9) marked softening and contraction of the spleen, with anemia; (10) slight atheromatosis of the abdominal aorta, and (11) status following an old cesarean section, with scar in the anterior wall of the uterus and ligation of the tubes; fibrous adhesions in the pelvis.

Chief Pathologic Diagnosis.—The chief pathologic diagnosis was ruptured aneurysm of the splenic artery with massive hemorrhage into the lesser sac and peritoneal cavity; generalized anemia.

Summary of Case.—The formation of the aneurysm may be attributed to an embolus arising from the heart valve. The depressed scarring of the left kidney may also be attributed to infarction arising from the same source, which was probably the so-called "pyelitis" of six months previous. There were no evidences of arteriosclerosis along the course of the splenic artery or in the wall of the aneurysm, although there was slight atheromatosis of the abdominal aorta. There was no evidence of syphilis. The lesion was of some duration, as was shown by indentation into the stomach, atrophy of the pancreas and lamination of the clot, but not long enough for calcification to have taken place.

Rupture probably occurred in two stages, the first starting as a slow leak about thirty-six hours before the patient was first examined. This was probably confined to the lesser sac and the gastrohepatic ligament. The second rupture, which gave rise to the massive, fatal hemorrhage, probably occurred about nine hours after the first examination, when the patient complained of sudden sharp pain followed by a definite change in appearance and condition.

Age and Sex Incidence.—Aneurysm of the branches of the abdominal aorta is considered unusual. In 1928, Thompson [1] collected 65 cases of aneurysm of the hepatic artery, and Singer [2] collected 40 cases of aneurysm of the renal artery. In 1924 Baumgartner and Thomas [3] collected 40 cases of aneurysm of the splenic artery. These cases represented the incidence in the previous fifty years, evidence of the fact that the condition is rare. In 1929, Anderson and Gray [4] collected 58 cases of aneurysm of the splenic artery and reported an additional case. Their report included most of the 27 cases reported by Bertrand and Clavel [5] in the same year. Since 1929, we have collected 24 additional cases, and we report another in this paper. Lindboe [6] collected pathologic reports made by Schroetter, Muller, Bosdorf and Emmerich, who, altogether, in 41,437 autopsies observed 554 abdominal aneurysms, of which only 21 were in the splenic artery, i. e., 0.05 per cent of the whole body material.

Most of the aneurysms occurred in the third decade of life. The aneurysms occurring in each of the fourth, fifth, sixth and seventh decades were almost as many. The difference in the numbers in different decades was not great enough to make the data on age incidence definite. In the reported cases in which the sex of the patient was stated, the condition occurred twice as frequently in females as in males.

PATHOLOGIC PICTURE

The pathologic features of this lesion after rupture are difficult to interpret anatomically. In the upper part of the abdomen, especially in the lesser sac, there is usually a poorly circumscribed mass of recent and organized clot with adhesion of the viscera. It is difficult to identify the lesion, and only after careful dissection can the various organs be separated. Bertrand and Clavel [5] have made an extensive study of the pathologic picture and have compared the aneurysm to an inflammatory tumor, which has created all around itself multiple and thick adhesions to all the organs of the region, namely, the stomach (posterior surface), pancreas, colon and spleen.

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Twenty-Five Cases of Aneurysm of the Splenic Artery

Case No.	Author and Reference	Age, Sex	Comment	Physical Examination	Preoperative Diagnosis	Treatment	Result	Pathologic Picture
1	Brockman [23]	69 F	Violent pain in left upper quadrant; vomited; desire to defecate; no result with enema	Shock; bruit in left upper quadrant; temperature 97; pulse 120	Ruptured splenic aneurysm (?); acute intestinal obstruction	Splenectomy	Recovery	Marked hemorrhage from splenic pedicle; vessels and structures ploughed up by hemorrhage
2	Brockman [24]	? M	Acute abdominal pain; treated expectantly and improved; two weeks later seized with severe pain, vomited, passed blood by bowel and died suddenly	None recorded	None recorded	Rest; morphine	Death	Aneurysm of splenic artery, ruptured secondarily into large bowel
3	Tixier, Baumgartner, Ronneux and Gadreau [28]	40 F	Repeated violent hematemesis; coma relieved with insulin	Mass in left hypochondrium; marked asthenia	Round calicified area in spleen	Splenectomy	Recovery?	Two large saccular aneurysms partially calcified and with small dilatations more or less aneurysm-like on the branchings of the artery
4	Boecker, P.: Beitrag zur hämorrhagischen Milznekrose und Milzruptur mit Verblutung in die Bauchhöhle infolge Thrombose eines Aneurysmas der A. lienalis, Zentralbl. f. Chir. **58**: 1375, 1931	50 M	Operated on for adenocarcinoma of stomach; 4th postoperative day sudden pain left shoulder; temperature and pulse rate normal; relief with enema; 5th postoperative day attempted to sit up; sudden collapse and death	None recorded	Pulmonary embolus	None for lesion of splenic artery	Death	Aneurysm 3 cm. from splenic hilum with fibrous tissue and calcification; hemorrhagic necrosis and rupture of spleen subsequent to postoperative thrombus
5	Lower and Farrell [22]	16 M	Paroxysms of severe abdominal pain for 8 years; tarry stools; some relief with morphine; onset followed whooping cough	Pale and undernourished; underweight; deformity and hypermotility of duodenum	Chronic pancreatitis	Removal of tumor with part of tail of pancreas	Recovery	Aneurysm of splenic artery; fibrosis of glandular pancreatic tissue
6	Lindboe [6]	53 F	Epigastric pain; dyspepsia; lassitude	Anemic; undernourished; pale and thin	Calcified aneurysm of splenic artery	Splenectomy with removal of aneurysm	Recovery	Aneurysm of splenic artery with hyaline degeneration and calcification
7	Mallet-Guy [25]	67 F	Mild "stomach ache"; sudden sharp severe pain left hypochondrium; condition worse; 3 days later, signs of internal hemorrhage	Pale; anuria; fainting spells; systolic blood pressure 90; large mass in left upper quadrant; dull to percussion; noticeable pulsation and murmur	Ruptured aneurysm of splenic artery	Splenectomy; removal of aneurysm and portion of tail of pancreas	Death	Hemorrhage only into retroperitoneal space; aneurysm of splenic artery 6 cm. from spleen; ruptured posteriorly
8	Pujals, L. O.: Sabre un	22	Pain in right wrist 2 weeks;	Tense abdomen, painful	Hemorrhagic periton-	Exploration; source	Death	False aneurysm of splenic

8	Pujals, L. O.: Sabre un caso de falso aneurisma de la arteria esplenica, Rev. Soc. argent. de biol. **8**: 355, 1932	22 F	Pain in right wrist 2 weeks; few days later, severe pain localized in left hypochondrium and groin	Tense abdomen, painful to palpation; vaginal examination showed fundi of sac filled out and uterus deviated	Hemorrhagic peritonitis with possible perforation of the uterus	Exploration; source of bleeding not determined	Death	False aneurysm of splenic artery because part of wall of artery was missing, and giant infarct of spleen
9	Heüveldop [19]	? F	Eight months pregnant; no prodromal symptoms; sudden and acute onset	None recorded	None recorded	Died before hospitalization	Death	Massive hemorrhage; ruptured splenic aneurysm, septic, possibly mycotic, 1 cm. from hilus
10	LeFevre and Pettis [10].....	78 M	Sudden nonradiating epigastric pain; "enlarged spleen" for 3 years; hypertension 3 years	Acutely ill; anemic; blood pressure 108/52; pulsating lemon-sized mass in left upper quadrant; bruit; enlarged spleen	Aneurysm of abdominal aorta; splenomegaly; bilateral inguinal hernia	Supportive; no operation	Death	Abdomen full of blood; ruptured aneurysm of splenic artery; patches of calcification present
11	A. A. Remizov [12].....	26 F	Colicky abdominal pain and bloody stools	Tenderness of left side of abdomen; tumor palpated under left costal arch; ascites	Dysentery; chronic ulcerative colitis	Splenectomy and removal of aneurysm	Death	Aneurysm in hilar region of enlarged spleen; thrombi in splenic veins
12	Sered and Steiner [9].....	30 F	Sudden severe epigastric pain; right shoulder pain; vomiting; full term pregnancy	Cyanotic; shallow respiration; uterus not contracting; tender in epigastrium; no shock	Ruptured viscus	Low cervical cesarean section	Death	Massive abdominal hemorrhage; ruptured splenic aneurysm; sclerosed intima
13	Rupture of a Saccular Aneurysm of the Splenic Artery, Cabot Case 21211, New England J. Med. **212**: 991, 1935	74 F	Twelve hours before admission, sudden upper abdominal pain and collapse; vomited 2-3 times during night	Low blood pressure on admission; acute distress; distended abdomen; tender lower quadrants, especially the left	None recorded	Exploratory laparotomy	Death	Massive abdominal hemorrhage; ruptured aneurysm of splenic artery about 1.5 cm. in diameter as it passes through tail of pancreas
14	Osborne [21]	43 F	Intermittent colicky pain in right upper quadrant, radiating to right shoulder; epigastric fulness; nausea and occasional vomiting; 27 years previously, gallbladder explored	Slight tenderness in right upper quadrant; gallbladder normal to roentgen examination	Cholecystitis with stones	Exploration; clamps on aneurysmal incision left in situ	Death	Aneurysm of splenic artery with calcification and laminated clot; gallbladder normal
15	Dahm, M.: Das Röntgenbild der verkalkten Milzarterie und eines verkalkten Aneurysmas der Milzarterie, Röntgenpraxis **8**: 802, 1936	? F	Nonreferable to splenic artery	None recorded	Empyema of gallbladder; multiple liver abscess	None directed to splenic lesion (condition discovered at autopsy)	Death	Cherry-sized aneurysm in center of splenic artery with thick calcified walls
16	Johan Haffner [26]	51 F	Diffuse vague abdominal discomfort for 10 years; pain worse when standing	Sensitive in epigastrium	Calcified aneurysm of splenic artery	Splenectomy and removal of aneurysm	Recovery	Aneurysm of splenic artery at hilus of spleen, 2 by 2 by 1.6 cm.; wall calcified; degenerative changes in the artery
17	Rudnev, N. M.: A Case of Splenomegaly with Aneurysm of the Splenic Artery, Sovet. khir., 1936, no. 3, p. 534	?	Original article not seen	.....	....	.....	.....	Aneurysm of splenic artery; splenomegaly

Twenty-Five Cases of Aneurysm of the Splenic Artery—Continued

Case No.	Author and Reference	Age, Sex	Comment	Physical Examination	Preoperative Diagnosis	Treatment	Result	Pathologic Picture
18	Säfwenberg [7]	66 F	None referable to splenic lesion	Signs referable to urinary tract	Carcinoma of bladder; aneurysm of splenic artery	None directed to splenic artery (condition discovered at autopsy)	Death	Carcinoma of bladder; 2 aneurysms of splenic artery 1.6 by 1.1 cm. and 1.1 by 0.8 cm.; spleen not enlarged
19	Säfwenberg [7]	68 F	Fatigue; bad appetite; loss of weight	Signs referable to urinary tract	Polycystic kidney; cholelithiasis; aneurysm of splenic artery	None directed to splenic artery (condition discovered at autopsy)	Death	Calcification of splenic artery; two aneurysms about 1 cm. in diameter
20	Parsons [20] (October 1934)..	45 M	Cramplike epigastric pain; vomiting; collapse with left shoulder pain; improved in 5 days; recurrence 6th day and hospitalized	Shock; abdomen distended, rigid and tender, especially left lumbar region	Perforated gastric ulcer	Splenectomy and removal of aneurysm	Recovery	Aneurysm of splenic artery; fibrous hyaline degeneration of vessels
21	Parsons [20] (same patient August 1936)	47 M	Severe upper abdominal pain; vomiting; readmitted	Shock; distended abdomen; tender left upper quadrant; dull in left loin	"Picture of a woman bleeding from ruptured ectopic gestation"	Cavity of aneurysm packed	Death	Massive hemorrhage; ruptured aneurysm of splenic artery prox. to point of removal in first operation
22	Cady [8]	50 F	None referable to splenic aneurysms	Acutely ill owing to widespread infection arising from other source; splenomegaly	Common duct obstruction; aneurysms seen at autopsy	None directed to splenic lesion	Death from acute infection	Seven aneurysms of the splenic artery, the largest 8 cm. and the smallest 1.5 cm. in diameter; calcification in walls
23	Schuster [11]	62 F	None referable to splenic aneurysm	Picture of congestive heart failure; severe anemia; familial hemorrhagic telangiectasis	.....	None directed to splenic aneurysm	Death from bronchopneumonia	Three aneurysms of splenic artery with calcified walls; patchy deficiency of media and internal elastic lamina; congenital
24	Fuchs [29]	60 M	Pain in upper part of abdomen; vomiting; constipation loss of 12 pounds (5.4 Kg.) in last 2 months	Not described	Gastric ulcer; aneurysm of splenic artery	Not recorded	Not stated	No confirmation of roentgen diagnosis reported
25	Machemer and Fuge..... (present paper)	30 F	Mild upper abdominal pain; urgency and frequency; pain more severe, especially in lower part of abdomen	Shock; marked pallor; exquisite tenderness and spasm in lower part of abdomen	Rheumatic heart disease; ruptured ectopic pregnancy on admission but subsequent course was much too severe; exsanguinating intra-abdominal hemorrhage, source unknown	Fluids; heat; morphine; died before transfusion could be given	Death	Aneurysm of splenic artery; rupture in superior and inferior part; filled with soft laminated mixed clot

The aneurysmal wall is usually thickened, surrounded by old clot in which it is difficult to find the fissure or rupture. The rupture itself is variable in size and location, ranging from a small punched-out area to an irregularly torn rip. In some instances considerable quantities of calcification are observed within the wall of the sac, while in others, as in our case, no calcification is present.

The splenic artery in some instances has been involved in generalized atherosclerosis, as described by Säfwenberg.[7] In Cady's case [8] the atherosclerosis was localized to the splenic artery. In Lindboe's case [6] fibrous degeneration was present, while in Sered and Steiner's [9] and LeFevre and Pettis' [10] cases sclerosis and calcification were present only in the media of the splenic artery.

In Schuster's case [11] the main trunk of the splenic artery showed a fine fibrosis and loss of elastic tissue of the media. In a secondary branch there was reduplication of the internal elastic lamina, while in the tertiary branch there was a complete deficiency of the media over the point of the cleft.

In 12 of the 27 cases reported by Bertrand and Clavel [5] the spleen was hypertrophied. Remizov [12] reported the presence of splenomegaly in about 50 per cent of cases of aneurysm of the splenic artery. Microscopic examination of the spleen showed only chronic passive congestion of the splenic parenchyma. In LeFevre and Pettis' case [10] splenomegaly was diagnosed three years prior to the occurrence of fatal hemorrhage from the ruptured splenic aneurysm.

ETIOLOGY

In this review, as in others, it seems impossible to mention an outstanding causative factor. Aside from varying degrees of calcification found in the wall of the aneurysm, atheromatous changes are most frequently seen in microscopic examination of the artery. Syphilis is said to be an etiologic factor, but in none of our cases was it present. In 6 cases of the series reported by Baumgartner and Thomas [3] it was mentioned particularly that there was no history of syphilis, and the

7. Säfwenberg, O.: Zwei röntgenologisch diagnostizierte Fälle von Milzarterienaneurysma, Acta radiol. **18**:481, 1937.

8. Cady, J. B.: Aneurysm of the Splenic Artery, Guthrie Clin. Bull. **6**:145, 1937.

9. Sered, H., and Steiner, L. M.: Full Term Pregnancy Complicated by Ruptured Splenic Aneurysm, Am. J. Obst. & Gynec. **29**:606, 1935.

10. LeFevre, G. L., and Pettis, E. M.: Aneurysm of Splenic Artery with Fatal Hemorrhage, J. Michigan M. Soc. **34**:358, 1935.

11. Schuster, N. H.: Familial Hemorrhagic Telangiectasia Associated with Multiple Aneurysms, J. Path. & Bact. **44**:29, 1937.

12. Remizov, A. A.: Saccular Aneurysm of the Splenic Artery, Sovet. khir., 1935, no. 8, p. 136.

findings and Wassermann reaction were negative. Binder [13] stated that syphilis is not a factor except in aneurysm of the aorta. Chronic infection and direct or indirect trauma have been considered etiologic factors in reported cases because symptoms shortly followed the infection or trauma.

Ponfick [14] stated that endocarditis is one of the main factors responsible for aneurysm of the splenic artery. He expressed the opinion that for the production of an aneurysm there must be an embolism at the branching of a vessel which lies in loose supporting tissue.

Tarrozzi [15] described an aneurysm of the splenic artery and noted the lack of elastic membrane. He thought that localized collections of elastic tissue with intervening areas without this tissue might be a causative factor.

In the splenic aneurysm described by Schuster [11] the internal elastic lamina showed short lengths of reduplication into poorly stained strands, which entirely disappeared in certain places. In one area there was a complete deficiency of the media, the gap being filled with connective and elastic tissue of the adventitia. Schuster stated that aneurysm of the splenic artery may be another manifestation of inborn vascular defects. Other arterial systems are subject to the same hazard of multiple aneurysmal dilatations, notably the cerebral, hepatic, renal and coronary arteries. In series of cases of aneurysm of these arterial systems, he stated, there are certain inexplicable cases in which the aneurysm might be regarded as having a congenital basis. The original suggestion he attributed to Eppinger.[16]

Selter [17] stated the opinion that increased blood pressure following embolism is essential to the formation of an aneurysm. Rolleston [18] added that a weakened vessel wall in addition to increased pressure is necessary for formation of an aneurysm. Remizov,[12] in a sense,

13. Binder, V.: Aneurysm der Arteria lienalis mit tödlicher Blutung, Verhandl. d. deutsch. path. Gesellsch. **16**:225, 1913.

14. Ponfick: Ueber embolische Aneurysmen, nebst Bemerkungen über das acute Herzaneurysma (Herzgeschwur), Virchows Arch. f. path. Anat. **58**:528, 1873.

15. Tarrozzi, G.: Ein echten Aneurysma der Milzarterie, Centralbl. f. allg. Path. u. path. Anat. **15**:700, 1904.

16. Eppinger, H.: Pathogenesis (Histogenesis und Aetiologie) der Aneurysmen einschliesslich des Aneurysma equi verminosum, Arch. f. klin. Chir. (supp.) **35**: 1, 1887.

17. Selter, P.: Ein Aneurysma der Milzarterie, entstanden in Folge einer durch Embolie hervorgerufenen Blutdrucksteigerung, Virchows Arch. f. path. Anat. **134**:189, 1893.

18. Rolleston, F.: Aneurysm of the Splenic Artery, Tr. Path. Soc. London **50**:55, 1898.

combined these two ideas in stating that there are two main factors in the development of an aneurysm of the splenic artery, preliminary degeneration of the arterial wall and a consequent or concomitant rise of blood pressure. In his case old thrombi were present in the splenic vein, which suggested stasis in the splenic circulation and increased pressure in the splenic artery.

While reviewing these various conceptions, it is interesting to note that 8 cases [19] of aneurysm of the splenic artery have been reported in which the condition appeared as a complication of pregnancy. In all cases it appeared during the eighth and ninth months or during labor. The condition was usually diagnosed as ruptured viscus. In all cases it proved to be a fatal complication of the pregnancy.

CLINICAL ASPECTS

In reviewing the clinical picture of reported cases, it is readily apparent that the greatest obstacle to diagnosis is the absence of a definite clinical picture. In the case reported by Parsons [20] the symptoms closely simulated those of gastric ulcer and perforation. In Osborne's case [21] the picture was that of cholecystitis with cholelithiasis. In Lower and Farrell's case [22] the clinical picture and findings were those of chronic pancreatitis. The pressure of the aneurysm caused extensive fibrosis of the glandular tissue, producing an external secretory deficiency, although the islet tissue remained intact. In many instances there was no history to suggest the presence of an abdominal lesion until hemorrhage occurred. Repeatedly in the female the diagnosis of "ruptured ectopic pregnancy" was made. In a few cases the lesion produced no symptoms and was found incidentally at autopsy.

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19. Wesenberg, W.: Verblutung während der Geburt infolge Ruptur einer Aneurysmas der Milzarterie, Zentralbl. f. Gynäk. **36**:463, 1912. Van Rooy, A. H. M. J.: Rupture of Splenic Aneurysm at End of Pregnancy, Nederl. maandschr. v. geneesk. **14**:507, 1927. Lundwall, K., and Godl, A.: Aneurysm of Splenic Artery Ruptured at the Ninth Month of Pregnancy with Fatal Hemorrhage, Arch. f. Gynäk. **113**:177, 1923. Saenger, H.: Fatal Hemorrhage in the Eighth Month of Pregnancy from Rupture of Aneurysm of Splenic Artery, Zentralbl. f. Gynäk. **50**:1324, 1926. Mayer, E.: Verblutung nach der Geburt infolge Ruptur eines Aneurysmas der Arteria lienalis, ibid. **52**:754, 1928. Remmelts, E.: Case of Sudden Death During Pregnancy from Rupture of Aneurysm of the Splenic Artery, Tijdschr. v. prakt. verlosk. **32**:126, 1928. Heüveldop: Ein Fall von Ruptur eines Aneurysmas der Milzarterie, Centralbl. f. allg. Path. u. path. Anat. **61**:277, 1934. Sered and Steiner.[9]

20. Parsons, C. G.: A Case of Ruptured Aneurysm of the Splenic Artery with Recurrence, Brit. J. Surg. **24**:708, 1937.

21. Osborne, S. E.: Aneurysm of the Splenic Artery Simulating Cholecystitis, Lancet **1**:1007, 1936.

22. Lower, W. E., and Farrell, J. I.: Aneurysm of the Splenic Artery: Report of a Case and Review of the Literature, Arch. Surg. **23**:182 (Aug.) 1931.

Aneurysm of the splenic artery may be considered a symptomless lesion until its effect on neighboring viscera or surrounding structures is manifest or until rupture occurs. Pain is the most common symptom at the onset and is usually located in the epigastrium. It is usually mild and may be colicky, although other types have been described. Parsons [20] stated that the pain of gastric ulcer is probably due to severe spasm of the muscular wall of the stomach. In his case the aneurysm was adherent to the lesser curvature of the stomach, and it is possible that by irritation it produced a similar type of spasm. Dyspepsia, weakness, lassitude, nausea and vomiting were occasionally present in the reported cases. In some cases there was an enlargement of the spleen or a mass was palpable in the upper part of the abdomen. Occasionally a pulsation was felt or a bruit was heard in the upper part of the abdomen. At the time when extensive rupture and massive hemorrhage occur there is violent pain quite different from the pain at onset.

Brockman [23] pointed out that rupture of the aneurysm takes place in two stages. The first rupture occurs in the lesser sac, producing mild peritonitis with subsequent formation of adhesions. The primary rupture is usually not fatal, since clotting in the more or less closed space of the lesser sac occurs in a short time. At some later time a secondary rupture occurs, with severe internal hemorrhage; this usually terminates fatally. In most cases the aneurysm ruptures secondarily into the abdominal cavity, although it has been known to rupture into the stomach, the colon, the stomach and colon, the stomach and abdominal cavity or (once) the splenic vein.

Bertrand and Clavel [5] expressed the opinion that the rupture occurs progressively. The wall of the aneurysmal pocket becomes fissured, creating all around it a hematoma, which tends to become organized, thus creating new adhesions. The first hemorrhage, therefore, occurs usually in a mass of adhesions, which tends to limit it. Bertrand and Clavel expressed the opinion that the evolutionary character of the entire aneurysmal and perianeurysmal mass determines the clinical aspects and contributes to the difficulty of diagnosis.

The clinical picture after secondary rupture is that of an acute condition of the abdomen. It may simulate and has been diagnosed as perforated gastric ulcer, acute intestinal obstruction, acute pancreatitis, pulmonary embolism, mesenteric thrombosis, ruptured ectopic gestation or ruptured viscus in pregnancy. The final clinical picture has usually been that of severe internal hemorrhage.

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23. Brockman, R. St. L.: Aneurysm of the Splenic Artery, Brit. J. Surg. **17**: 692, 1930.

DIAGNOSIS

As has been stated, the diagnosis has rarely been made before operation. Högler [24] has in 2 cases diagnosed aneurysm of the splenic artery on the basis of a systolic murmur over the hilus of the spleen. In both cases the diagnosis was verified at autopsy. Brockman [23] heard a bruit in the left upper quadrant of the abdomen which suggested to him an aneurysm of the splenic artery, but because of the clinical picture he felt that the condition was acute intestinal obstruction. Brockman stressed the value of abdominal auscultation in diagnosis of this lesion. Mallet-Guy [25] found a large mass in the left upper quadrant of the abdomen, which was dull to percussion and had a noticeable pulsation. A murmur was heard over the mass, and a diagnosis of ruptured aneurysm of the splenic artery was made. The diagnosis was confirmed by operation.

Lindboe [6] reported a case in which symptoms caused the patient to seek aid before rupture occurred and the diagnosis was made by roentgen examination. A sharply defined calcareous ring was found behind the stomach. Repeated roentgen examinations ruled out an aneurysm of the adjacent arteries, principally the renal and gastric sinistra. The diagnosis of aneurysm of the splenic artery was confirmed by operation.

Haffner [26] reported 1 case and Säfwenberg [7] 2 cases in which the condition was diagnosed by the roentgen findings. Round, irregular shadows of calcification were seen in the left upper quadrant of the abdomen. The calcareous areas were proved to be outside of the stomach and the kidney. The shadows moved on respiration and were less dense in the center. Haffner's diagnosis was confirmed by operation, and Säfwenberg's diagnoses were confirmed by autopsy.

Israelski [27] described a case in which a twisted shadow with double contour was seen with the aid of the roentgen rays. The diagnosis, although suggested, was not definitely made until autopsy, when a calcified splenic artery with cylindric dilatation of the middle part was found.

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24. Högler, F.: Beitrag zur Klinik des Leber- und Milzarterienaneurysmas, Wien. Arch. f. inn. Med. **1**:509, 1920.

25. Mallet-Guy, P.: Anévrisme de l'artère splénique rompu dans l'arrièrecavité des épiploons et le tissu cellulaire rétropéritonéal, Arch. franco-belges de chir. **33**:1064, 1932.

26. Haffner, J.: Fall von verkalktem Aneurysma der Art. lienalis, Acta radiol. **17**:602, 1936.

27. Israelski, M.: Die verkalkte Arteria lienalis im Röntgenbilde, Röntgenpraxis **2**:670, 1930.

Tixier, Baumgartner, Ronneux and Gadreau [28] reported a case in which a shadow was seen on roentgen examination but a definite diagnosis was not made until operation. The difficulty they encountered in diagnosis was attributed to a 90 degree rotation of the spleen on its vertical axis, which cast the shadow in the position of the splenic parenchyma.

Fuchs [29] reported a case of aneurysm of the splenic artery in which the lesion was diagnosed by roentgen examination. There was a walnut-sized shadow of calcification below the left side of the diaphragm, near the midline, which moved with the diaphragm. No confirmation of the diagnosis, however, is reported.

PROGNOSIS AND TREATMENT

Lower and Farrell [22] collected from the literature 15 cases in which some surgical procedure was attempted. Seven of the patients recovered, an operative mortality of 53 per cent. In 4 cases a tampon was used to control the bleeding, and all the patients died. In 1 successful case the large vessels entering and leaving the aneurysm were ligated. All other successes followed removal of the aneurysm, the spleen and, in 2 instances, a portion of the adjacent pancreas.

In the series reported here, 18 patients died, a general mortality of 75 per cent. Thirteen of the patients were operated on, and 7 died, an operative mortality of 47 per cent. However, in only 8 of the 13 operative cases was an attempt made to remove the aneurysm and the spleen or to ligate the vessels. In this group of 8 cases, 2 patients died, an operative mortality of 25 per cent. In the other unsuccessful cases, such procedures as packing the cavity, clamping the sac and simple exploration were done.

Obviously the treatment of this lesion is entirely surgical, although the exact diagnosis of its type and location cannot always be made before operation. The ideal treatment consists of removing the aneurysm, the spleen and, if need be, the adjacent pancreas. The value of heat, morphine, fluids and adequate transfusion should not be overlooked. Packing the marsupialized cavity and clamping the sac are only palliative procedures, which at the moment may save the patient, but require further surgical procedures for a permanent cure.

Dr. Samuel Sanes of the Department of Pathology furnished the pathologic description in the case presented.

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28. Tixier, Baumgartner, Ronneux and Gadreau: Anévrismes calcifiés de l'artère splénique et splénomégalie, Bull. et mém. Soc. de radiol. méd. de France **18**:349, 1930.

29. Fuchs, G.: Das Röntgenbild des Aneurysmas der Arteria lienalis, Röentgenpraxis **9**:467, 1937.

HYPERFUNCTIONING ADENOMA OF AN ECTOPIC PARATHYROID GLAND

REPORT OF A CASE

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DETROIT

HISTORICAL CONSIDERATIONS

The parathyroid glands, which are derived from the third and fourth branchial clefts,[1] were first recognized and named by Sandström in 1880. A parathyroid tumor was first recognized by de Santi in 1900. Gradually the relation between such a tumor and the syndrome of von Recklinghausen's disease was realized, and in 1925 Mandl [2] reported the cure of the latter by removal of an enlarged parathyroid gland. Since then, reports of about 200 cases have been added to the medical literature.

ANATOMY

The parathyroid glands are present in all animals down to fishes. They may be situated on, in or behind the thyroid gland but are most frequently found on its posterior aspect, near the point at which the inferior thyroid artery enters the gland.[3] Rarely, aberrant or accessory parathyroid glands have been reported as present in the thymus or in the anterior mediastinum.[4] Millzner [5] has frequently found parathyroid glands on the anterior surface of the thyroid, but Gilmour [1] found only two anteriorly situated in 428 dissections. Four parathyroid glands are usually present, but careful search sometimes reveals only three or even two. Gilmour found an average of four parathyroids in each subject but actually found four in 87 per cent, two in 0.2 per cent, three in 6.1 per cent, five in 6 per cent and six in 0.5 per cent. More, up to

1. Gilmour, J. R.: The Gross Anatomy of the Parathyroid Glands, J. Path. & Bact. **46**:133, 1938.

2. Mandl, F.: Klinisches und Experimentelles zur Fragen der lokalisierten und generalisierten Ostitis fibrosa, Arch. f. klin. Chir. **143**:245, 1926.

3. Lahey, F. H., and Haggard, G. E.: Hyperparathyroidism, Surg., Gynec. & Obst. **60**:1033, 1935.

4. Hunter, D., and Turnbull, H. M.: Hyperparathyroidism: Generalized Osteitis Fibrosa, with Observations upon Bones, Parathyroid Tumors and Normal Parathyroid Glands, Brit. J. Surg. **19**:203, 1931.

5. Millzner, R. J.: The Occurrence of Parathyroids on the Anterior Surface of the Thyroid Gland, J. A. M. A. **88**:1053 (April 2) 1927.

eleven and twelve, have been reported, but there is some doubt as to the accuracy of the reports.[1] The parathyroids are shaped like lima beans. They are reddish or light brown. Their maximum size is 8 mm. in length by 4 mm. in width.

PATHOLOGY

Hyperparathyroidism is a disease due to hyperplasia of the parathyroid parenchyma or to the presence of a hyperfunctioning adenoma resulting in increased secretory activity of the gland.[6] In diffuse hyperplasia only a portion of one gland (rarely, portions of two glands) may be involved.

Adenomas of the parathyroid glands are smooth and firm. They are round or ovoid and may vary greatly in size. As a rule they are small, being rarely palpable. Sometimes they are not more than twice the size of a normal gland and yet produce symptoms. Growths have been reported, however, which weighed 300 Gm. or more. The most typical cell of such neoplasms is a large clear or vacuolated cell, the *wasserhelle* cell, which is similar to the typical clear cell of hypernephroma. Smaller, acidophilic cells also may be present.[6a] Hyperparathyroidism is due to hyperfunctioning adenoma seven or eight times as frequently as to hyperplasia.

The secretory hyperactivity of the gland produces a calcium-phosphorus imbalance, causes migration of calcium from bone and results in hypercalcemia. Calcium and phosphorus in the blood are subject to the laws of ionic dissociation; that is, the concentration of calcium ions and that of phosphate ions if altered must vary inversely with each other in order that they may remain in equilibrium with the amount of undissolved calcium phosphate.[7] The first action of the parathyroid hormone is to sweep phosphates from the blood into the urine. The phosphate content of the serum then falls, and consequently the calcium content rises. The excess of calcium is secreted by the kidneys, and the reserves of both calcium and phosphate are mobilized from bone. The loss of calcium salts from bone produces a lesion of the skeletal system known as osteitis fibrosa cystica or von Recklinghausen's disease.

The osseous changes consist of decalcification, the formation of degenerative cysts, hemorrhagic extravasation and replacement of the decalcified bone by connective tissue containing numerous giant cells of the osteoclastic type. These giant cells may be fused phagocytes the function of which is to remove osseous debris. Pathologic fractures are common. Hemorrhage is part of the picture of active decalcification

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6. Castleman, B., and Mallory, T. B.: The Parathyroids in Hyperparathyroidism, Am. J. Path. **11**:1, 1935.

6a. Warren, S., and Morgan, J. R. E.: The Parathyroid Glands: A Histologic Study of Parathyroid Adenoma, Arch. Path. **20**:823 (Dec.) 1935.

7. Taylor, H.: Osteitis Fibrosa: An Experimental Study, Brit. J. Surg. **22**:561, 1935.

because the high concentration of calcium damages the vascular endothelium.[7] Hemorrhage interferes with healing.

Because of hypercalcemia the calcium concentration of the urine is increased, and the formation of urinary calculi is a prominent part of the disease. Uremia may result from impaction of the renal pelvis with crystals of calcium phosphate.[8] A case in which death occurred from such uremia was observed by one of us (O. A. B.).

CLINICAL FEATURES

Hyperparathyroidism occurs two and one-half times as frequently in females as in males. While cases have been reported in which the condition occurred from the second to the ninth decade, in about half of

Fig. 1.—Operative scar, indicating the site of the tumor.

all cases it occurs in persons between 40 and 60 years of age. In only 6 per cent of reported cases has it occurred in the second decade.

Clinical symptoms [8a] usually consist of pain and localized swelling of bone, possibly with deformity and disturbance of gait. Nephrolithiasis and associated renal infection may lead to abdominal symptoms sometimes diagnosed as duodenal ulcer or appendicitis. Polyuria and polydipsia may be present. Terminally the patient may lose height owing to destruction of the skeletal system. However, hyperparathyroidism is

8. Elson, K. A.; Wood, F. C., and Ravdin, I. S.: Hyperparathyroidism with Renal Insufficiency, Am. J. M. Sc. **191**:49, 1936.

8a. Gutman, A. B.; Swenson, P. C., and Parsons, W. B.: Differential Diagnosis of Hyperparathyroidism, J. A. M. A. **103**:87 (July 14) 1934.

not necessarily associated with osseous changes. Many patients with hyperparathyroidism have no symptoms referable to the skeletal system and present no roentgen evidence of disease of bone. There may be no elevation of the phosphatase content of the blood and no evidence of osseous changes at biopsy.[9]

It is generally understood that the disease is characterized by a high calcium and a low phosphorus content of the serum. Shelling[10] stated that the lower limit of the value for serum calcium in cases of hyperparathyroidism is 12.5 mg. per hundred cubic centimeters. However, Albright and his associates[9] stated that the average value for serum calcium was below 12.5 mg. in 40 per cent of their 35 cases, the lowest average being 10.7 mg. Chemical examination of the urine reveals a high urinary output of calcium, and the total protein content of the serum is low.

Roentgen findings consist of osteoporosis and the presence of cysts.[11] The calvarium may be thickened and granular and the tables of the skull indistinct. The cortex of the long bones may be thin, indistinct and irregular. All the bones may be involved. Renal calculi may be demonstrated.

In the differential diagnosis of the osseous lesions associated with hyperparathyroidism the following diseases must be considered:

Focal osteitis fibrosa
Metastatic carcinoma
Multiple myeloma
Osteogenesis imperfecta
Single bone cyst
Senile osteoporosis
Paget's disease
Osteomalacia

Space does not permit a detailed discussion of the differential diagnosis. The reader is referred to the recent literature.

TREATMENT

Surgical removal of the adenoma or the involved gland results in immediate correction of the disturbance in calcium-phosphorus metabo-

9. Albright, F.; Sulkowitch, H. W., and Bloomberg, E.: Further Experience in Diagnosis of Hyperparathyroidism, Am. J. M. Sc. **193**:800, 1937.

10. Shelling, D. H.: The Parathyroids in Health and Disease, St. Louis, C. V. Mosby Company, 1935.

11. Camp, J. D.: Osseous Changes in Hyperparathyroidism: A Roentgenologic Study, J. A. M. A. **99**:1913 (Dec. 3) 1932.

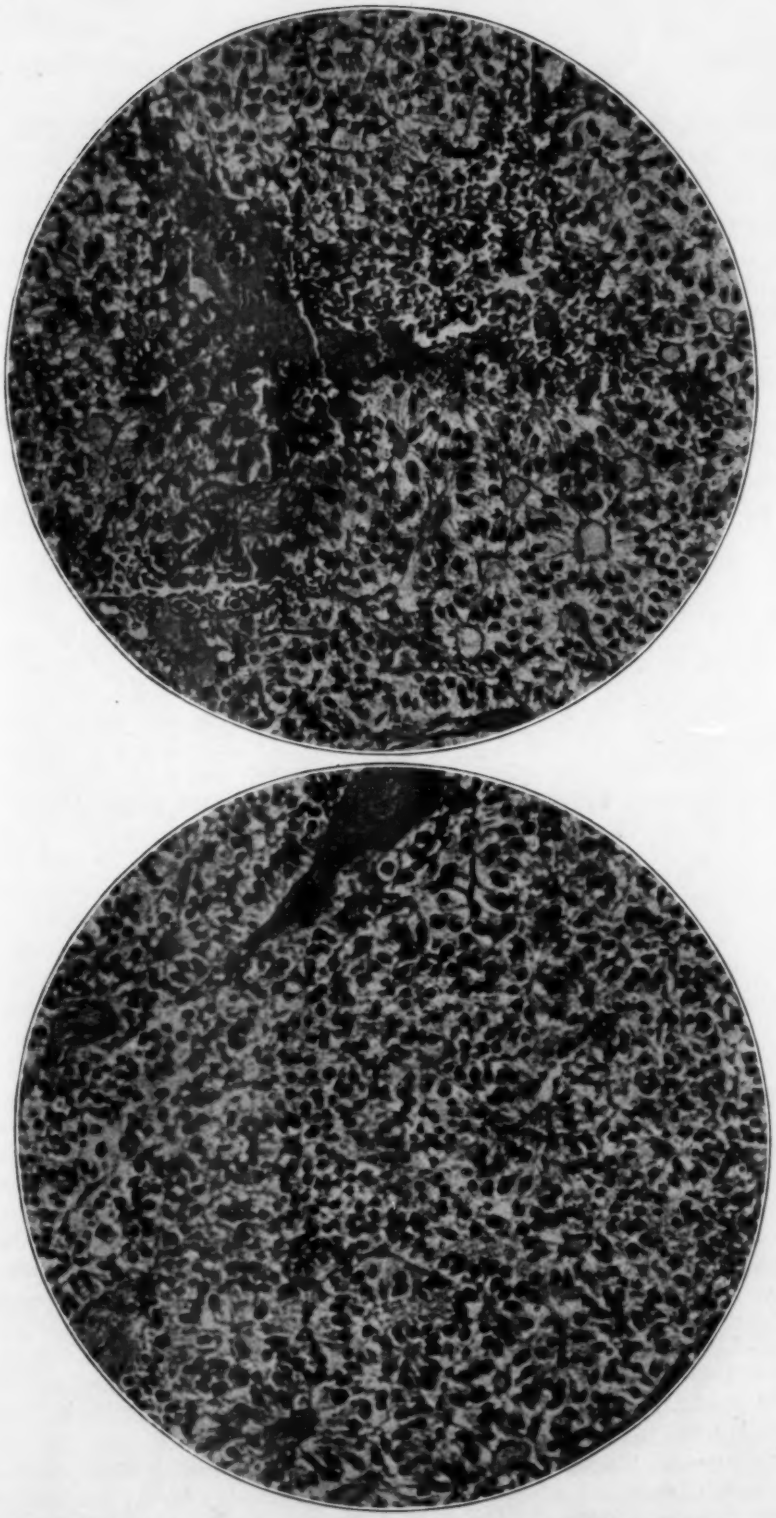

Fig. 2.—Photomicrographs representing two different portions of the adenoma.

lism. Improvement in many symptoms usually follows. If the disease is in an early stage and the osseous changes are mild, a complete return to normal structurally may be expected. In cases of more advanced involvement repair of bone occurs slowly, and in cases of far advanced involvement complete recovery cannot be expected. Immediate symptomatic and structural improvement is impressive, but the results are not necessarily permanent. Location of the adenoma or hyperplastic gland at operation is not always easy, and multiple operations have been necessary, 1 case having been reported in which the adenoma was found at the seventh operation.[12] Recurrence of symptoms due to the formation of other adenomas or to hyperplasia occurring in remaining glands have been reported, necessitating reoperation. Other cases have not been followed for long periods. Roentgen therapy may decrease the activity of the gland, but experience with this form of treatment has been limited.[13] Administration of calcium, phosphorus and vitamin D has been advocated.[14]

REPORT OF A CASE

M. S., an 18 year old white girl, was first seen in the office of one of us (V. L. B.) on Feb. 1, 1936, complaining of recurrent pain in the right lower quadrant of the abdomen and of two tender nodules in the right anterior triangle of the neck. Neither complaint was disabling, and she appeared for examination because her sister had recently had a ruptured appendix. Her past history was entirely irrelevant. Her mother had been confined to a sanatorium for tuberculous patients for one year and had been discharged with a healed lesion at the apex of the right lung three years previously. The two lumps in the patient's neck had been present for six months. Two lower teeth on the right side had been extracted on the advice of a physician. The abdominal pain was characteristic of mild chronic appendicitis. The patient had lost about 5 pounds (2.3 Kg.) in weight during the past six months. The menstrual history was normal. There was no history of arthritis, fractures or pains in the joints.

Physical Examination.—The tonsils had been removed in childhood. Examination of the eyes, ears, nose and throat otherwise gave negative results. The thyroid gland was palpable. Just below and to the right of the cricoid cartilage there was a spherical tumor 2.5 cm. in diameter, which appeared to be rather deeply seated in the neck but was freely movable and not attached to the skin. There was a somewhat smaller but tender mass in the submaxillary area. Examination of the chest gave negative results. There was abdominal tenderness at McBurney's point. Rectal examination revealed a small uterus. There was no adnexal tenderness. The ovaries were not palpable. Examination of the extremities gave entirely

12. Churchill, E. D., and Cope, O.: Parathyroid Tumors Associated with Hyperparathyroidism, Surg., Gynec. & Obst. **58**:255, 1934.

13. Cutler, M., and Owens, S. E.: Irradiation of the Parathyroids in Osteitis Fibrosis Cystica, Surg., Gynec. & Obst. **59**:81, 1934.

14. Albright, F.; Aub, J. C., and Bauer, W.: Hyperparathyroidism: A Common and Polymorphic Condition as Illustrated by Seventeen Proved Cases from One Clinic, J. A. M. A. **102**:1276 (April 21) 1934.

negative results. Examination of the blood revealed the following values: hemoglobin, 90 per cent; erythrocytes, 4,400,000 per cubic millimeter and leukocytes, 7,500 per cubic millimeter, with polymorphonuclear neutrophils 62 per cent, lymphocytes 36 per cent and eosinophils 2 per cent. The coagulation time was three and one-half minutes. Urinalysis gave negative results.

Progress.—The patient was admitted to the hospital on February 14. A diagnosis of mild chronic appendicitis and cervical lymphadenitis of undetermined type was made at the time of admission. Appendectomy was performed. The appendix was normal on gross examination. The abdomen was thoroughly explored, but no abnormality was found. A submaxillary lymph node measuring 15 mm. in

Fig. 3.—Roentgenograms of the lower end of the right femur, *A* taken on Oct. 8, 1936, and *B* on Dec. 5, 1938. They show almost complete filling in of the cyst.

diameter was removed. The larger tumor in the neck was situated beneath the platysma muscle, lying on the lower portion of the right lobe of the thyroid gland at its lateral border. This tumor was removed without difficulty. The postoperative convalescence was uneventful. The patient was discharged from the hospital on the sixth postoperative day.

Pathologic Report (O. A. B.).—The appendix was normal. The cervical lymph node exhibited chronic inflammatory hyperplasia. The remainder of the specimen consisted of an ovoid encapsulated mass measuring 23 mm. in maximum diameter. It was rather soft and friable. On section it was homogeneous, opaque and

diffusely pink. Microscopic sections consisted entirely of an epithelial neoplasm which was apparently encapsulated. The individual neoplastic cells were columnar and fairly large and possessed clear or faintly staining cytoplasm. There was some irregularity in the size of the cells; some areas were composed of smaller cells which were somewhat basophilic. In the larger cell areas there was a definite arrangement into cords or tubules, with recognizable lumens in some instances. Throughout the tumor there was a rich vascular stroma, with conspicuous engorgement of blood vessels in some areas. The pathologic diagnosis was hyperfunctioning adenoma of a parathyroid gland.

Roentgen Report (Dr. R. W. McGeoch).—There was considerable difficulty in persuading the patient to return for further observation, and roentgen examination was not made until May 2, two and one-half months after the operation. Roentgenograms of the skull, hands, shoulders, knees and spine were taken. There was a fine granular mottling throughout all the bones, with marked irregularity and thinning of the trabeculae. The skull had a moth-eaten appearance, with small areas of increased density. There was a cyst in the distal extremity of the fifth metacarpal bone of the right hand, one at the upper end of the left humerus on the medial side just below the epiphysial line, one in the lower end of the right femur above the medial condyle and one at the upper end of the left tibia on the medial side. The roentgen diagnosis was osteitis fibrosa cystica.

Fig. 4.—Roentgenograms of the upper end of the left humerus, *A*, taken on Oct. 8, 1936, and *B* on Dec. 5, 1938.

Further Data.—The first opportunity to examine the blood chemically was on May 2, two and one-half months after operation. At that time the calcium content of the blood was 10.5 Gm. per hundred cubic centimeters. The patient had no complaints, no disabilities and no symptoms referable to the extremities. Her posture was good. She received no further treatment. On October 8 she had no

complaints and physical examination gave negative results. She had gained 10 pounds (4.5 Kg.) in weight in the past two years. Pain in the right lower quadrant of the abdomen had not recurred. On Dec. 5, 1938, a check-up roentgen examination of the right hand, right knee and left shoulder revealed almost complete filling in of the cysts which were found in the distal end of the fifth metacarpal bone of the right hand, in the upper end of the left humerus and in the lower end of the right femur in the original examination. The trabeculations were still irregular and thickened and showed a tendency toward cyst formation. In a comparison of the roentgenograms with those taken on Oct. 8, 1936, greater density in the trabeculations of the cyst was apparent.

COMMENT

The sequence of diagnostic and therapeutic events in this case was irregular, because the correct diagnosis was not suspected until the tumor had been removed and a pathologic report rendered. For this reason chemical examination of the blood was not made until too late to be of any value, and roentgen examination of the skeletal system was delayed for the same reason. A clinical diagnosis of tuberculous cervical adenitis was made because (1) there were two masses in the neck and (2) there was a history of contact with tuberculosis. Pathologic examination revealed that the smaller lump in the neck was a hyperplastic lymph node. The cause of the hyperplasia was undetermined.

The unusual features of this case were as follows: 1. The patient was in the second decade of life, in which only 6 per cent of parathyroid adenomas occur. 2. The tumor was palpable. 3. Excision was performed easily, in contrast to the difficulty frequently encountered in locating the tumor. 4. The adenoma apparently developed in an ectopic parathyroid gland. 5. The patient was cured of the primary disease before the correct diagnosis was suspected clinically.

The osseous lesions demonstrated roentgenographically had almost completely disappeared within eight months after the operation, and the patient was entirely well in the interval. At the time of writing, after nearly three years, there is no clinical or roentgen evidence of recurrence. The symptoms leading to a clinical diagnosis of appendicitis were unexplained except that small renal calculi might have been present. Examination of the right kidney, however, did not reveal the presence of calculi.

SIMPLE STANDARD APPARATUS FOR TREATMENT OF COMPOUND FRACTURES OF THE HAND, FINGERS AND WRIST

REPORT OF A CASE AND EVALUATION OF THE END RESULT

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NEW YORK

The literature abounds with descriptions of all types of apparatus which have been of benefit in specific cases of fracture or severe injury of the hand and fingers. There is, however, no uniformly recognized method of making these appliances practical for all kinds of injuries to the hand and fingers. Moreover, I know of no appliance that can be said to be universally easy to procure when needed nor any that is as simple and safe to use as the one which is herein described.

The best therapy is often the most simple to prescribe and carry out for the really complicated compound hand injuries mentioned. The various principles of treatment of compound fractures of the hand and fingers need only to be mentioned in order to demonstrate the simplicity and ease of quick construction of my appliance.

The types of case in which the method applies are varied. It may be used for any or all of the following injuries:

1. Compound fracture of one or all fingers.
2. Compound fracture of the hand and/or the thumb.
3. Compound fracture of the wrist and/or fracture of the hand and/or fingers.

The cost of materials for this apparatus is difficult to estimate, but it is negligible. The materials (fig. 1) include the following:

1. Heavy wire-cutting pliers.
2. Several steel wire coat hangers.
3. Plaster of paris bandages (2, 3 and 4 inch [5, 7.5 and 10 cm.] rolls, as used in standard hospitals).
4. Medium and small elastic bands (assorted), about one dozen.
5. Round, straight cambric sewing needles (assorted sizes).
6. Adhesive tape.

7. Six to twelve small corks.

8. Heavy suture silk, such as is kept in a standard hospital operating room supply.

9. Rolls of cotton batting and assorted gauze dressings and roll gauze bandages.

10. Iron extension ring—a "banjo splint," or a substitute as described.

The method of constructing the apparatus depends somewhat on the type and extent of the injury, as well as on the time after the injury when the patient is first seen.

No digression from the individual surgeon's principles of treatment of compound fractures need be made. The apparatus is compatible

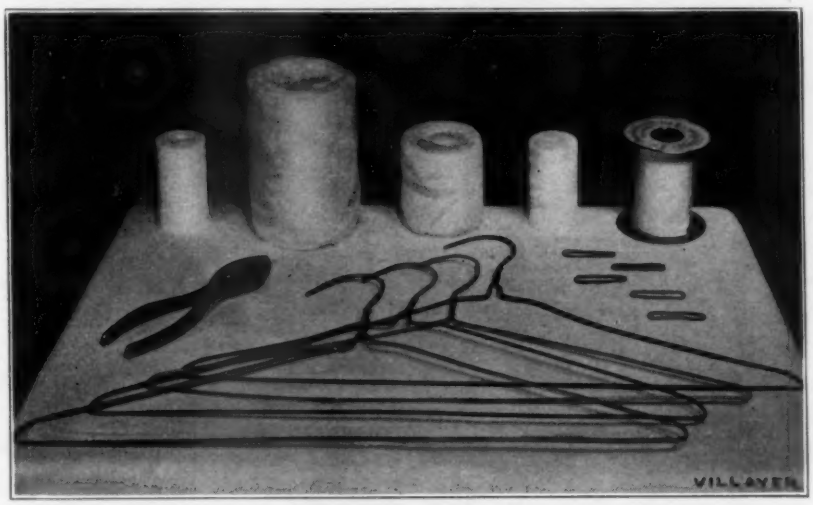

Fig. 1.—Essentials for construction of the apparatus: gauze rolls, padding, plaster of paris, adhesive tape, elastic bands, steel wire coat hangers and wire-cutting pliers. Sewing needles, corks and silk suture material are not shown.

with either "open" or "closed" treatment and may be adapted to either. It causes no shock in application, and in cases in which the patient is brought to the hospital in profound shock it may be put on without the slightest harm provided its limitations are understood. It will then be in position, so that the surgeon may later apply traction in any way he sees fit, according to the roentgen indications.

Before entering on a description of the apparatus it is important to emphasize the main principles of the treatment of compound fractures. After these are in mind it will be seen that the method of treatment to be described complies exactly with these principles. Careful consideration must be given each case before any apparatus is con-

demned or accepted. The mechanism advocated in this communication, I believe, will withstand the most critical observation of specialists in the treatment of complicated fractures of the hand. The idea of the apparatus is probably not entirely original; the appliance is a combination of several kinds of apparatus, which have their own separate merits. One must learn how to use it after it is in place and must try it out to see its simplicity of action and its general practicability. It is certainly a reliable mechanism for making the treatment of severe injuries of the hand and fingers and even of the wrist a business-like and clear-cut standard procedure for every hospital.

The main principles in the treatment of all compound fractures can be briefly reviewed in a few words: First, the risk of infection must be minimized (regardless of the type of treatment used, whether "open" or "closed"). Second, satisfactory reduction must be aimed at even if it cannot always be achieved. Third, immobilization must be obtained and maintained at the direction of the surgeon and not be left to chance. Fourth, traction must be obtainable when necessary and if necessary must be uniformly maintained. Fifth, changes in the direction of traction must be possible with whatever apparatus is used, so that it may be possible to overcome a probably bad start in the treatment of the more severe and shocking accidental injuries. Sixth, early mobility should be possible. Seventh, the need of visibility and accessibility of the wounds of compound fractures can hardly be stressed too much when a mechanical aid is required, especially with injuries to the hand. Lastly, the patient's comfort must be considered from beginning to end if a good result is to be obtained. All these provisions can be carried out if the apparatus I describe is used wisely and modified to the needs of the individual patient.

One or two other suggestions about the treatment of compound fractures seem so obvious that they need only be mentioned to insure their not being forgotten or neglected. Shock and hemorrhage must at all times be the first considerations in the treatment of such injuries. One reason is that the hand and fingers have a large nerve supply, predisposing to more shock than is often supposed, and another is that the arteries of the hand and wrist are large and can be the cause of marked loss of blood, sometimes out of proportion to the visible injury.

One point to stress in the construction of a standard apparatus for fractures of the hand and finger with severely contused wounds is the proper application of one of the parts of the apparatus I shall describe. I refer to the plaster of paris portion of the mechanism. No plaster should be applied to any part of any extremity unless the part to be covered is clean and one is positive that no active or potentially dangerous infection is being covered.

Plaster, then, in this apparatus must be thought of only as an agent to fix the extremity above the fractures, the wounds and the entire length of the broken bones (except those of the forearm in some cases) being left free and clear to be later immobilized by an entirely different means.

The plaster will serve only as a firm, safe foundation for other parts of the apparatus. The likelihood of applying plaster to an arm which may later become infected will be minimized if the plaster is not applied too tightly, if the operator has had experience with its handling and makes sure that plenty of cotton batting is used before the plaster is rolled on.

When a hand is badly mangled up to the wrist, I know of no better way of treating the injury than by this method, but provision must be made not to endanger the forearm from constriction below a safe point, some inches above the wrist. The elbow should be flexed and

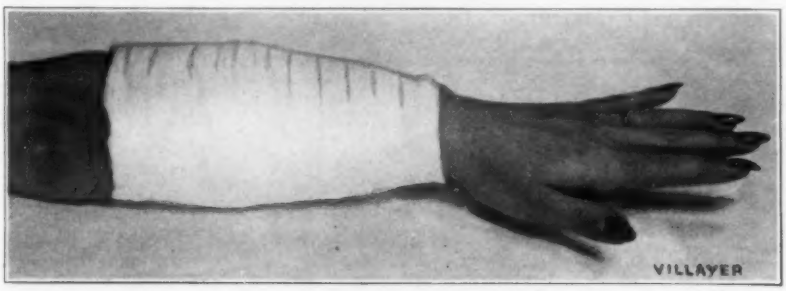

Fig. 2.—Application of plaster of paris between the wrist and the elbow, over ample padding, with sufficient plaster to make a firm foundation for the rest of the apparatus after this much has been allowed to harden.

the plaster cast carried halfway to the shoulder on the upper part of the arm in such a case.

Since a hint as to the procedure has been given, the general structure of the apparatus may be discussed. Figure 2 illustrates the points where the plaster is first applied.

The plaster may ordinarily stop short of the elbow by a sufficient margin to insure comfortable flexion and should extend to the wrist, where plenty of padding should be used before the plaster is put on.

This first application should be the thickness of a cast that will be solid but not bulky, usually requiring at least three to four rolls of 3 or 4 inch plaster. The plaster should be allowed to harden. Time will vary for this, but it is most important that this foundation cast be firm, so that later applications of iron arms or the steel wire will not dent the plaster and possibly cut off the circulation or cause pressure necrosis.

The next step is to cut the flexible steel wire coat hangers so that plenty of straight pieces may be obtained. A straight length of at least a foot and a half (45 cm.) when bent so as to form the general shape of a banjo head and handle will suffice to make the banjo splint extension ring that so often is hard to find when needed. If the wire from two more coat hangers is wrapped around this framework in the manner of a grapevine winding around a small branch of a tree, the banjo arm will be strong and inflexible. This assures a fixed radiating surface distal to the hand for the application of traction in the line of the forearm, as is shown in figure 3. If the iron banjo splint is available it may be utilized, of course, in place of the coat hanger wire.

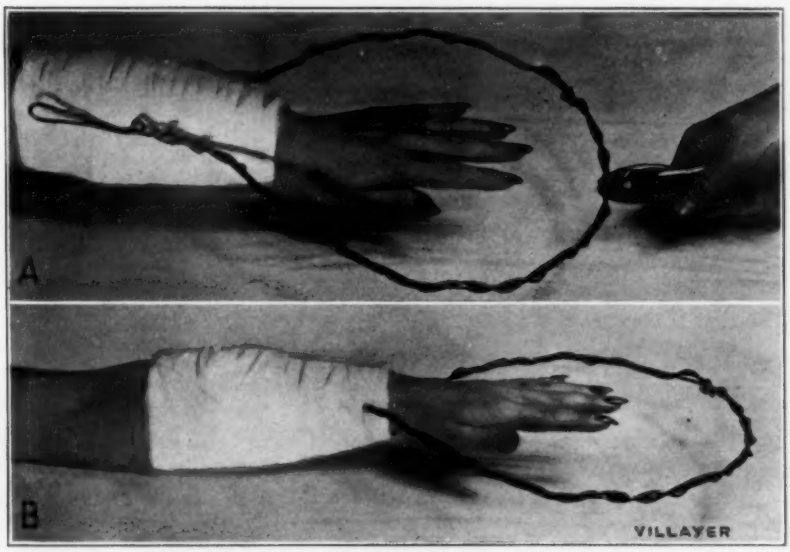

Fig. 3.—*A*, coat hanger wire bent in the shape of a banjo splint and reenforced by two more lengths of the same material, wound around it like a grapevine. This makes a strong "banjo splint" in a few moments. *B*, banjo splint incorporated into place by the addition of a few turns of wet plaster over the now solid foundation cast.

Next, it is important to decide just what kind of extension of the hand itself is needed and what amount of flexion of the hand and fingers seems indicated.

The procedure may be varied to suit the requirements of the given case. Two other modified "banjo arms" made of the coat hanger wire can be wrapped into the cast after fixation of the main banjo extension arm by one or two rolls of plaster. The shape of these two additional arms is illustrated in figure 4.

The apparatus is nearly finished and requires only the fixation which will be given by stabilization of the upper and lower wire arms. This is carried out by attaching their distal corners to measured lengths of the coat hanger wire (about 8 cm., with 1 cm. ends bent at an angle of about 45 degrees), by means of a few turns of adhesive tape, as shown by figure 5. This completes the framework of the apparatus.

A clear description of the apparatus is difficult, but it is unbelievably simple to understand when it is tried on a patient.

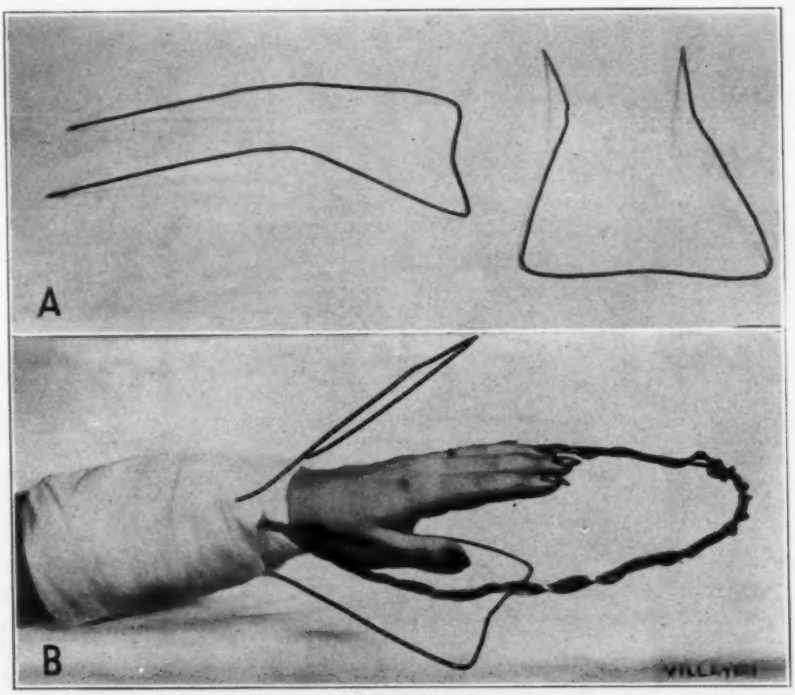

Fig. 4.—*A*, additional pieces of coat hanger wire bent into these shapes so as to furnish the two upper and lower modified "banjo arms." *B*, upper and lower "modified banjo arms" held in place by a few more turns of plaster over the foundation cast.

Shortening of the upper and lower arms is easy and is accomplished by the addition of cross wires fixed at right angles in the same way as the "fixation wires" by bending their ends, followed by application of a few turns of adhesive tape.

An idea of the bare framework of the apparatus (without all the attachments to the fingers and countertraction attachments afforded by the use of muslin bandage around the fingers and extending over the side arms) is gained from simplified model photographs taken at a different angle (fig. 6).

In a photograph the "upper" and "lower" arms of the steel coat hanger wire would be partly obscured by the turns of adhesive tape which hold the fixation wires in place and by the muslin bandage tied around the fingers so as to provide countertraction when necessary, in the direction of either the "upper" or the "lower" cross arm; therefore, no attachments of any sort are shown in the model pictures. The small snapshots in figure 7 show the actual apparatus as used in the case herein reported.

Little further explanation is necessary to illustrate how easily the wounds on the dorsal or palmar side (or both) of the hand and fingers

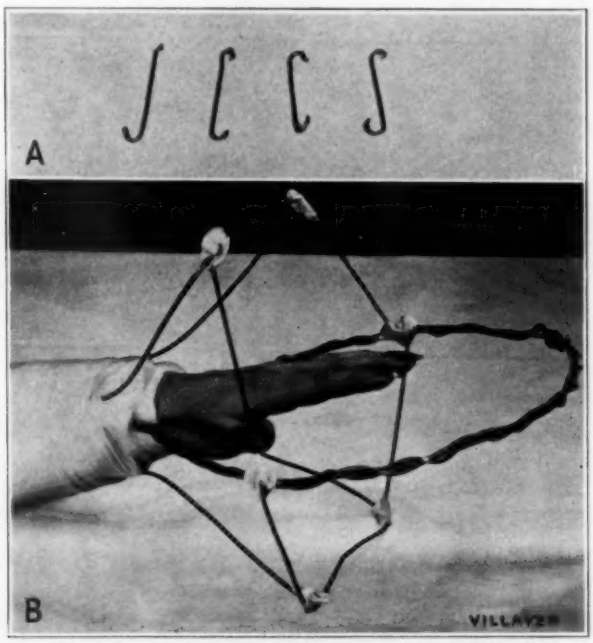

Fig. 5.—*A*, lengths of coat hanger wire, about 8 cm. long with 1 cm. ends bent at an angle of 45 degrees, make satisfactory side arms with which to stabilize the upper and lower "modified banjo arms" by attaching them to the main banjo splint. *B*, bare framework of the apparatus completed by attachment of the 8 cm. lengths of wire from the upper and lower arms to the main banjo splint by adhesive tape. It is easy now to see how attachments can be utilized for extension and counterextension in almost any direction.

can be reached or to show how extension and countertraction in all directions can be obtained by using rubber bands, attaching them by an interposed silk suture either to a finger nail or to the ends of a needle inserted through the fleshy tip of the finger, as shown in figure 8, or by adding extra cross wires to change the line of pull (not shown).

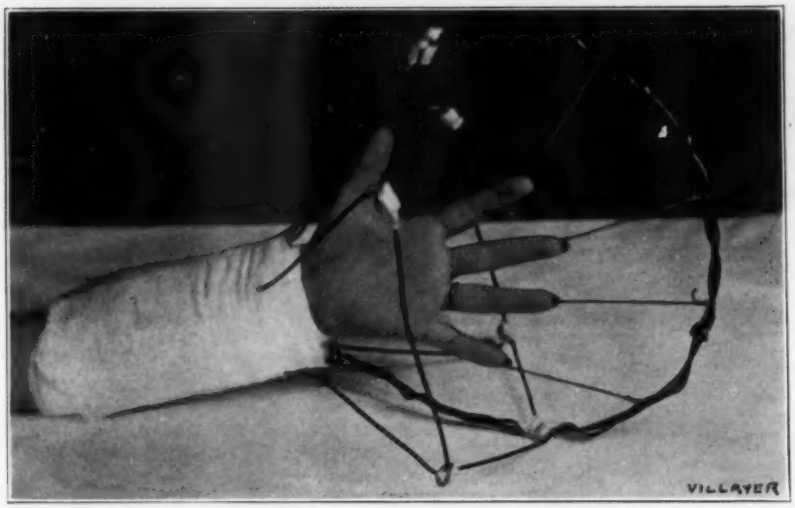

Fig. 6.—Bare framework of the apparatus in another view, with elastic bands connecting the model's finger nails to the main banjo splint. Actually the finger nails may be used for extension by threading them with silk suture material and attaching the silk to the elastic and, in turn, the elastic to the banjo splint. Counter-extensions by bandages connected with the upper and lower arms are not illustrated in this photograph, but may be seen in figure 7.

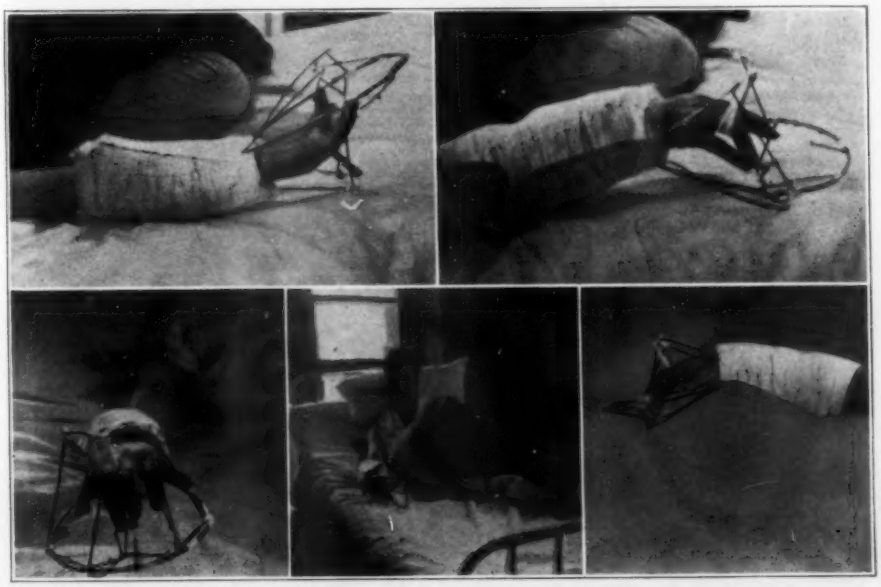

Fig. 7.—Views of the patient whose case is here reported, illustrating the mobility of the hand and the counterextension utilized on the ring finger by a muslin sling connected to the upper arm of the apparatus. The details of extension for the fingers are difficult to see, but the hand and fingers are held in almost balanced traction by elastic bands attached to the ends of the fingers (either by a silk suture through the finger nail or by a needle through the fleshy tip of the finger, as shown in figure 8).

Another type of cross arm, well padded and carefully bent into place, may be applied to the base or dorsum of the wrist, as shown in figures 9 and 10, if the cast cannot be applied as far distally as the wrist because of lacerations or fractures near the bases of the metacarpal bones.

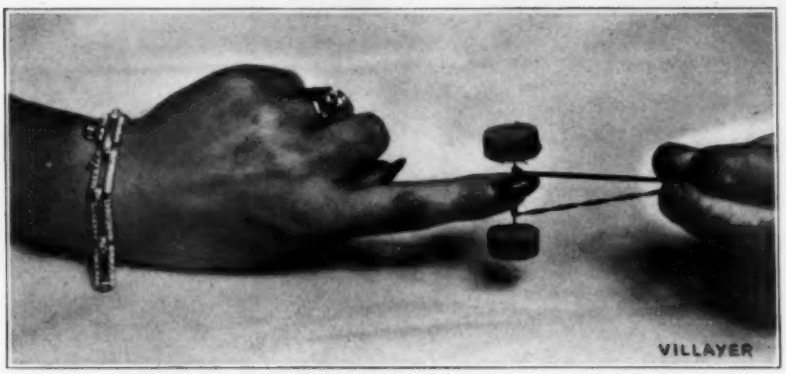

Fig. 8.—Method of attaching the elastic rubber band to the needle which is inserted through the fleshy tip of the finger. Protection of other fingers is provided by corks covering the ends of the needle.

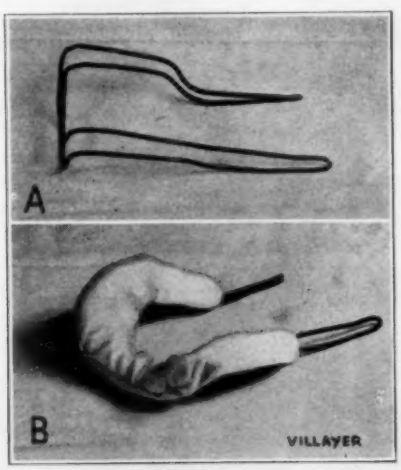

Fig. 9.—*A*, another piece of coat hanger wire, properly bent as shown. This makes an ideal support which can be added to the foundation cast if because of extensive lacerations on the flexor side of the wrist the foundation cast cannot extend far enough distally to support the wrist. *B*, same, padded and wrapped with gauze.

It is seen, then, that the method depends on the needs of the patient and on the physician's understanding of what is required for the treatment of all the different kinds of fractures of the hand and fingers. Briefly, there is no other way than to study the needs of each separate

fracture, as to displacement, applicability of extension, applicability of counterextension and general applicability of the apparatus.

In the case here reported, for example, all the fingers of the right hand were involved, and it was found that one of the compound fractures of the ring finger was not satisfactorily reduced by comparatively straight extension during the first few days. It was simple to change the direction of pull after a day or two so that the finger was flexed at the joint between the first and second phalanges and pull in two directions exerted so as to overcome the likelihood of further

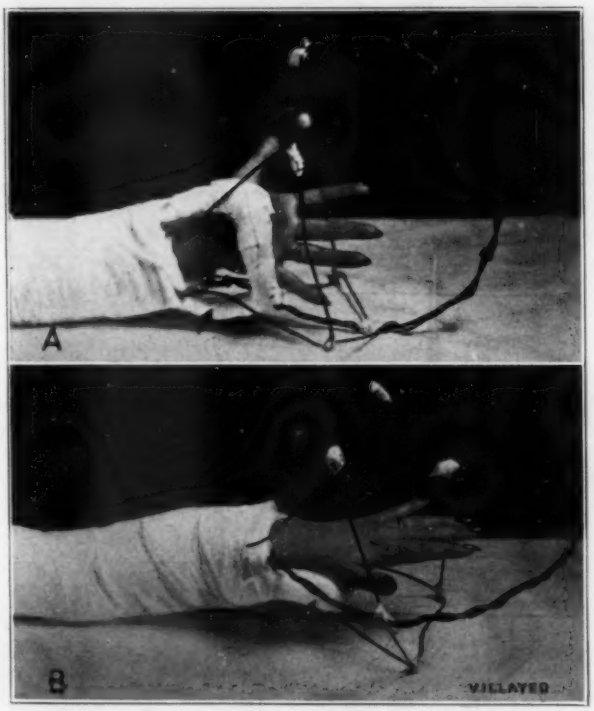

Fig. 10.—*A*, same general apparatus. The supporting arm shown in figure 9 is in place and is indicated by the arrow. *B*, different view, showing the supporting arm (indicated by the arrow) well padded and incorporated under the hand. A few more turns of plaster have been wrapped around it and around the foundation cast above the wrist.

displacement and to provide better reduction of the fracture of the middle phalanx.

The wounds on the dorsum of all the fingers, as well as those on the palmar side, were simple to dress, but the dressing would have been most difficult if any other kind of splint had been used to rest the fingers on or if the splint had had to be moved daily.

The case to be reported illustrates the simplicity and advantage of treatment with this apparatus. A detailed description of the injuries and final photographs of the hand six months after the injury, showing about 85 per cent return of function (about 75 per cent if considered cosmetically alone) are given. There was no complicating infection.

REPORT OF CASE

L. C., a man aged 20, a feeder on a dye cutter, was first seen at the New York Post-Graduate Medical School and Hospital on May 27, 1937, about twenty minutes after having caught all the fingers of the right hand in a dye cutter. He described the injury as a crushing one, but added that the cutter allowed about 1 cm. of clearance at most, so that part of the injury was due to his instinctive attempt to extricate his hand as it was being mashed.

General physical examination gave essentially negative results except for a fairly rapid pulse rate (about 100) and some degree of shock; there were pallor, weakness, sweating and nervousness. The urine was normal.

Both hands showed considerable discoloration with printer's ink. All the fingers of the right hand were blood stained, and had obviously undergone a severe crushing injury. It was difficult to tell which fingers were most injured. The middle finger was bleeding the least and appeared almost necrotic at first sight. The patient was not closely examined further but was admitted to the hospital after roentgen examination and was sent to the main operating room. No further treatment or examination was done preoperatively. A loose sterile gauze bandage was applied to the hand.

Operation (with the patient under nitrous oxide–ether anesthesia) consisted in careful sterilization of the hand and forearm with tincture of iodine and débridement of the edges of skin along some of the lacerations. Sterile saline solution was used to wash the depths of the wounds. Purified petroleum benzine, alcohol, saline solution and a repetition of the application of iodine were used where grease and dirt were adherent. A small bit of adhesive tape was removed from one of the fingers where the patient had had a small cut a few days before the present injury.

Examination was done in the operating room *at this time*, and not before. The roentgenograms were now available, and the patient was not subjected to any traumatizing handling of the fingers further than that needed to clean the wounds and make sure that no important ligaments or tendons were neglected.

Many lacerations were present, best portrayed by the shaded areas shown on the photographs in figure 11.

Roentgenograms demonstrated comminuted fractures of the midphalanges of the index, middle and ring fingers. There was avulsion of the extensor tendon of the middle finger at the first interphalangeal joint. The corresponding joint of the index finger and the distal joint of the extensor side of the little finger showed compound fractures, were lacerated and lying open. There were dislocation of the terminal phalanx and an avulsion of the nail of the little finger, which also showed a compound fracture.

It should be repeated that no blood was encountered on exploration of the badly injured middle finger. Hemostasis was obtained in all the other fingers, and all the wounds were partly closed with a total of only about five or six fine silk sutures. No flexor tendons were lacerated, although all were exposed on the palmar side in all the fingers. The tears in the capsules of the joints were not

repaired, nor was the inch or so of extensor tendon of the middle finger replaced. Instead it was cut off cleanly, because of its devitalized appearance.

Silk suture material was inserted through the nails of the index and middle fingers. Cambric sewing needles were used through the fleshy part of the ring and little fingers. The ends of the needles were covered by corks (fig. 8).

Next, an apparatus consisting of the cast previously described and a cast iron "banjo extension ring" splint was used. A similar mechanism has been illustrated in the photographs. Figure 7 shows the one actually used.

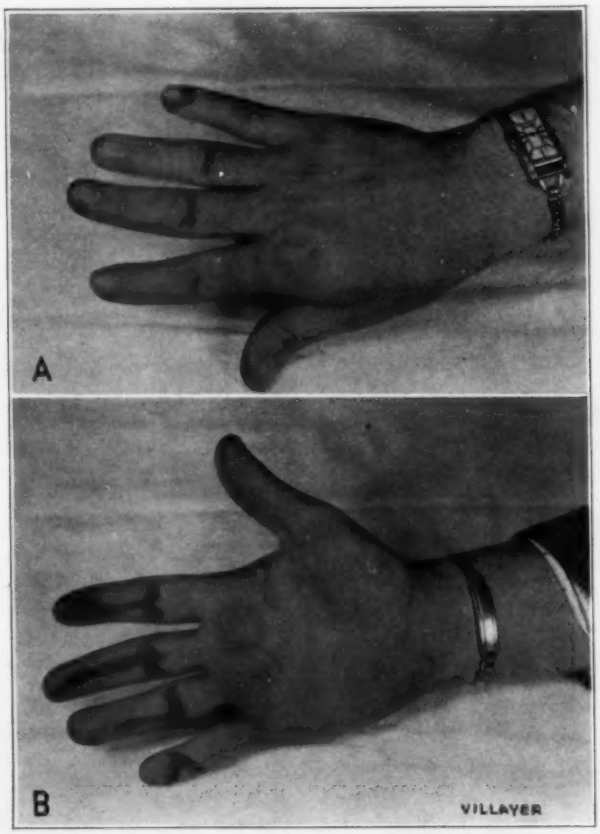

Fig. 11.—*A*, print of the left hand reversed, so that it appears to be the right hand. The shaded areas illustrate the lacerations on the dorsal side of the fingers, where compounded joint lacerations and compound fractures coexisted. Compare with the final result eight months after injury (figs. 13, 14 and 15). *B*, areas of laceration and cutaneous involvement over the exposed joints, fractures and deep tendons on the flexor side. Compare with the final result eight months after the accident, shown in figures 13, 14 and 15.

The fingers were moved little or not at all, to minimize trauma, and extension was held lightly by the silk sutures to the iron ring distally. No attempt was made to exert elastic tension on the fingers at this time because of the severity of the injuries and the danger of further loss of circulation with the onset of post-operative edema and congestion.

On the next day, when it became certain that circulation would continue satisfactorily and that amputation of any of the fingers would probably not be required, the apparatus was augmented by elastic band extension on the fingers.

Postoperative roentgen examination was thought less important for a few days than care of the wounds. No method of splinting the fingers could have been more satisfactory than the method used.

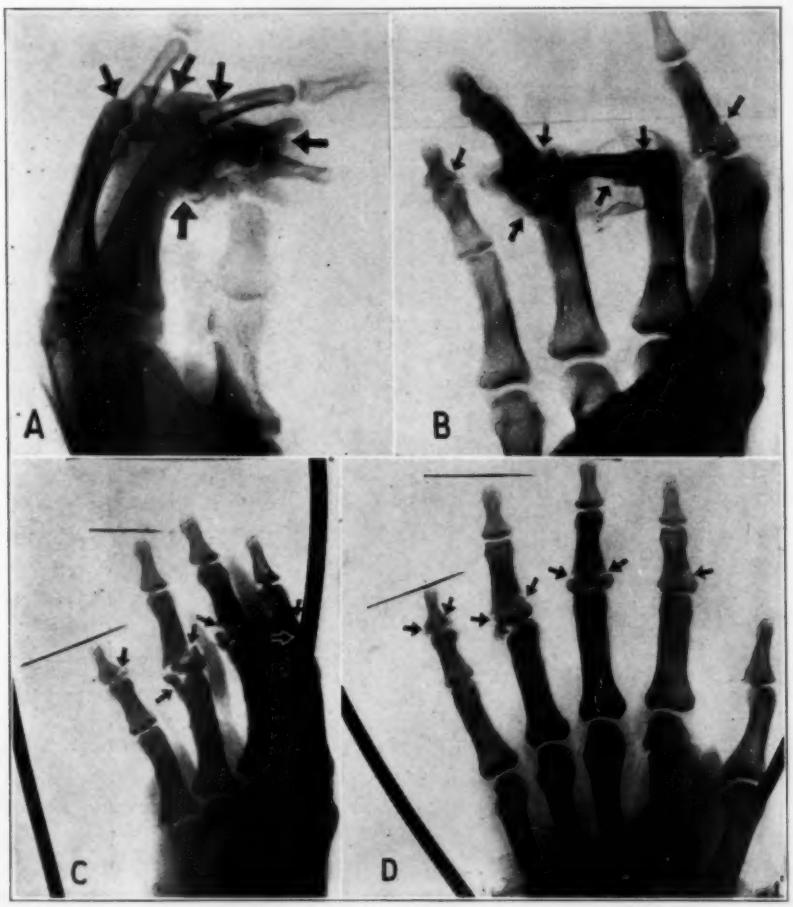

Fig. 12.—*A*, oblique fracture on the external side of the base of the midphalanx of the index finger. There is much capsular and soft tissue swelling. *B*, comminuted fracture of the base of the midphalanx of the middle finger, with a vertical fissure involving the articular surface. Swelling of the capsule and of the soft tissues is present. *C*, comminuted fracture of the midphalanx of the ring finger, with a vertical fissure line involving the articular surface; fracture of the mesial border of the head of the proximal phalanx, and slight palmar displacement of the major distal fragment of the midphalanx. There is considerable capsular and soft tissue swelling. *D*, comminuted fracture at the base of the terminal phalanx of the little finger, partly intra-articular, with capsular and soft tissue swelling.

When healing seemed to have begun, roentgenograms were taken, and all the fractures except that of the ring finger seemed to be fairly well reduced. It was at this time (about the fifth day) that the additional wire "arms" were added to the cast and braced with the stabilizing "fixation" wires of the same material, so that the apparatus could be utilized in a different way for one of the fingers, the ring finger. The application of a muslin bandage around the proximal phalanx of the ring finger and a change in the pull of the rubber extension band provided the difference in therapy indicated by the roentgenogram. Further displacement of the proximal fragment of the middle phalanx anteriorly was thus prevented,

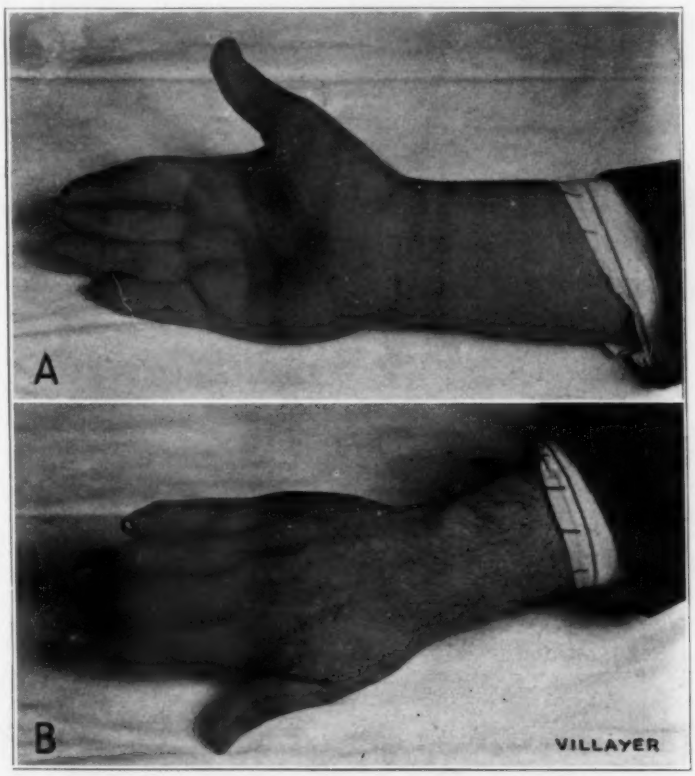

Fig. 13.—*A*, result eight months after the accident. *B*, same, viewed from the dorsal side.

and the chance of better reducing the distal fragment of the middle phalanx was increased. The mechanism succeeded fairly well and could have been easily modified again had it been found necessary.

By the third postoperative day the patient was encouraged to move the fingers slightly and not to mind the dressings, which consisted of careful removal of crusted blood and serum. At the time of the dressings the fingers were gently massaged toward the hand by means of small peroxide sponges. The patient volunteered the statement that the hand always felt better after the dressings. He had no pain and required sedatives only for a night or two.

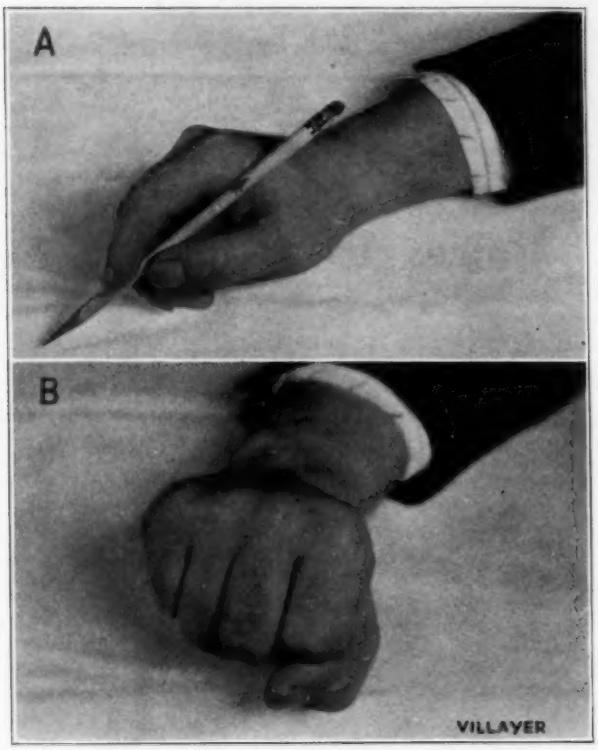

Fig. 14.—*A*, final result viewed from the side as the patient writes. *B*, final result showing a view of the fist the patient can make.

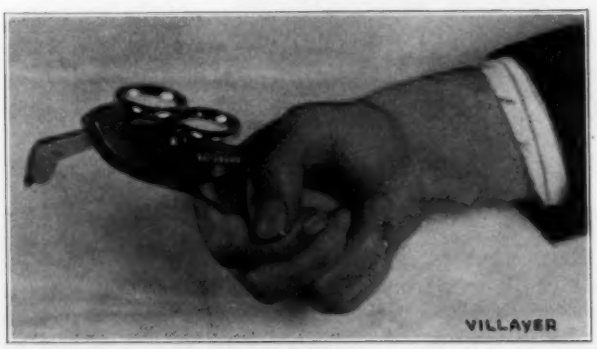

Fig. 15.—Final result showing the patient's ability to use tools eight months after the accident.

Shock played a part in this case, in that the patient could eat little and drink less for the first three days after the operation. Dextrose in saline solution was given intravenously on two occasions to counteract the gastric disturbances and compensate for the patient's inability to take in enough by mouth to offset the postoperative reaction and dehydration.

No infection occurred, partly owing to the use of strict aseptic technic from beginning to end. The wounds remained healed except for slight necrosis at the tip of the middle finger, at the base of the nail, for about two weeks. Culture of the exudate was sterile at the end of seventy-two hours.

The fingers were released from the apparatus at the end of four weeks, and the patient was instructed in detail about exercises and baths thereafter. Baking and massage were instituted in his daily regimen.

What the patient required more than anything else was repeated encouragement and example as to the type of exercise and the means of increasing the usefulness of the hand. It was difficult to convince him of the need of continuous effort over a period of months.

About nine weeks after the accident there was approximately 70 per cent return of function. At the time of writing, the cosmetic result is better than fair. There is some loss of soft tissue at the tip of the middle finger. There is inability to extend the middle finger completely at the distal two joints. The joints between the proximal and middle phalanges of the index, middle and ring fingers, all of which were badly lacerated, have remained somewhat larger in circumference than normal. The terminal joint of the little finger healed, and a new nail appeared, fortunately without any deformity to speak of.

The end result is illustrated by photographs taken eight months after the accident (figs. 13, 14 and 15).

[NOTE.—On April 20, 1939, nearly two years after the operation, the patient appeared to have suffered no retrogressive changes and had not the least complaint, the functional end result having remained the same as was observed seven months after the operation.]

SUMMARY AND CONCLUSIONS

Much has been written about special mechanisms and apparatus of all kinds for treatment of fractures of the hand and fingers, but little about simplification of the treatment of compound fractures in the same location, although this is a problem of vast importance to industrial surgeons and surgeons dealing with traumatic conditions, as well as to practitioners everywhere.

A definitely inexpensive, efficient and easy means of treating such injuries, particularly the worst lacerations and compound fractures, with especial regard to apparatus, is described in sufficient detail to recommend consideration of its adoption in almost all cases under nearly all hospital conditions. The materials required can be found in even the less expensively equipped institutions at a moment's notice.

A short review of the rules of treatment of compound fractures is included. The most important considerations in the treatment of compound fractures of the fingers and the hand are listed.

The case of a patient who suffered a mangling injury of all four fingers of the right hand, with several compound fractures, compound joint lacerations, avulsion of a tendon and lacerations of both sides of the fingers is described in detail up to eight months after the injury. The case illustrates the ease and efficacy of the management of such a severe injury with this apparatus. The estimated percentage of return of function is higher than might be expected from the use of other splints or mechanical aids.

There is a real need of better recognition of the importance of severe injuries of the hand and fingers and the best basic means of treating them. I believe I have shown a method which is possible, safe, efficient, inexpensive and uniform. It may be employed under different conditions and in various localities and is available to all physicians everywhere.

525 Park Avenue.

CALCIFICATION OF THE SUPRASPINATUS TENDON

CAUSE, PATHOLOGIC PICTURE AND RELATION TO THE SCALENUS ANTICUS SYNDROME

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A pathologic condition of the shoulder with a calcareous deposit which casts a shadow roentgenographically has become an increasingly popular subject since it was first described by Painter [1] in 1907. Nevertheless, failure to correlate the cause, the pathologic picture and the pathogenesis with the symptoms seems to be rather general.

This article is an attempt to correlate the pathologic lesion with the symptoms produced and to rationalize the treatment.

INCIDENCE

Calcareous deposits about the shoulder are much more common than is generally believed. In reporting 200 cases of "periarthritis of the shoulder," Dickson [2] found that 33.3 per cent showed a calcified deposit as revealed by the roentgenogram. Carnett,[3a] by routinely taking roentgenograms of both shoulders, disclosed that one fourth of his patients had bilateral deposits, only one shoulder being symptomatic at the time of examination.

Since the adoption about one year ago of a new routine for roentgen examination of the shoulder, a diagnosis of calcification of the supraspinatus tendon has been made in a series of 27 patients, 9 of whom had bilateral deposits. One additional patient had a calcareous deposit in the subscapularis tendon.

The occurrence of such deposits is uncommon before the thirtieth or after the fiftieth year of life. The youngest patient in my series

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1. Painter, C. F.: Subdeltoid Bursitis, Boston M. & S. J. **156**:345-349, 1907.

2. Dickson, J. A., and Crosby, E. H.: Periarthritis of the Shoulder: An Analysis of Two Hundred Cases, J. A. M. A. **99**:2252-2257 (Dec. 31) 1932.

3. (*a*) Carnett, J. B.: The Calcareous Deposits of So-Called Calcifying Subacromial Bursitis, Surg., Gynec. & Obst. **41**:404-421, 1925; So-Called "Subacromial Bursitis," S. Clin. North America **10**:1309-1317, 1930; So-Called Calcifying Subacromial Bursitis, Radiology **17**:505-513, 1931. (*b*) Carnett, J. B., and Case, E. A.: A Clinical and Pathological Discussion of So-Called Subacromial Bursitis, S. Clin. North America **9**:1107-1126, 1929.

was 31 and the oldest 68. Men usually are affected more often than women (2 to 1), but in the 28 recent cases 18 of the patients were females.

ANATOMY

Codman [4] in 1906 published the first adequate description of the subacromial bursa. He has since emphasized its mechanical importance and its relation to pathologic changes in the supraspinatus tendon.

The capsule of the shoulder joint in its superior portion blends with and becomes indistinguishable from the conjoined tendon of the short rotators as they course to their insertion into the tuberosities of the humerus. The tendon of the supraspinatus muscle reenforces the central portion of the capsule and is inserted into the anterior and uppermost part of the greater tuberosity. This attachment is just posterior to the bicipital groove, which may be palpated 2 fingerbreadths lateral to a line drawn vertically upward from the center of the cubital fossa when the elbow is flexed to a right angle.

The thin synovial lining of the subacromial bursa is tightly adherent to the tuberosities of the humerus and to the adjacent part of the conjoined tendons near their insertion to form its base and to the under surface of the acromion and adjacent structures to form its roof. On the whole, the bursa is circular, concavoconvex and somewhat smaller than the palm of the patient's hand, extending below the edge of the acromion as much as 1½ inches (3.7 cm.) at its lowest point. It is separated from the shoulder joint only by the conjoined tendons of the short rotators.

ETIOLOGY

Concerning the cause of calcareous deposits about the shoulder, it is fairly well agreed that the sequence of changes leading up to the deposition of calcium is primarily interference with the blood supply. There is considerable controversy, however, as to how these changes are brought about. Codman and Wright, some time after the publication

4. Codman, E. A.: On Stiff and Painful Shoulders: The Anatomy of the Subdeltoid or Subacromial Bursa and Its Clinical Importance; Subdeltoid Bursitis, Boston M. & S. J. **154**:613-620, 1906; Subacromial Bursitis, or Peri-Arthritis of the Shoulder Joint, ibid. **159**:533-537, 576-582 and 756-759, 1908; On Stiff and Painful Shoulders as Explaining Subacromial Bursitis and Partial Rupture of the Supraspinatus, ibid. **165**:115-120, 1911; Abduction of the Shoulder: An Interesting Observation in Connection with Subacromial Bursitis and Rupture of the Tendon of the Supraspinatus, ibid. **166**:890-891, 1912; Obscure Lesions of the Shoulder: Rupture of the Supraspinatus Tendon, ibid. **196**:381-387, 1927; Rupture of the Supraspinatus Tendon, Surg., Gynec. & Obst. **52**:578-586, 1931; The Shoulder: Rupture of the Supraspinatus Tendon and Other Lesions In or About the Subacromial Bursa, Boston, Thomas Todd Company, 1934. Codman, E. A., and Akerson, I. B.: The Pathology Associated with Rupture of the Supraspinatus Tendon, Ann. Surg. **93**:348-359, 1931.

of the first reports of rupture of the supraspinatus tendon, advanced the hypothesis that calcium is laid down in the unabsorbed hemorrhage which fills the defect in an abortive attempt at repair of minor injuries to the tendon tissue, which normally has a poor blood supply. Moschocowitz [5] and Elmslie [6] arrived at a similar conclusion. Carnett [8] recorded his opinion that the deposits are, as a rule, quiescent in their formation and are due to tendinitis, local necrosis of the tendon and calcification produced by often repeated occupational traumas which squeeze the supraspinatus tendon between the tuberosity of the humerus and the roof of the subacromial bursa. Brickner advanced the hypothesis of a metabolic factor, but his theory was not convincing.

In discussing pathologic calcification in general, Wells [7] concluded as follows:

> Any area of dead tissue that is not infected, and that is so large or so situated that it cannot be absorbed, probably will become infiltrated with lime salts. Most frequently calcified, next to totally necrotic tissues, are masses of scar tissue that have become hyaline subsequent to the shutting off of circulation in the scar by contraction of the tissue about the vessels. The calcium salts come from the blood where they are held in solution or in suspension by the proteins of the plasma in an unstable condition capable of being overthrown by the increased alkalinity of the blood resulting from changes in the carbon dioxide content. In the areas that are to become calcified, the circulation is very feeble, the blood plasma seeping through the tissues as through any dead or foreign substance of similar structure without the presence of red corpuscles to permit of oxidative changes, and the consequent production of carbon dioxide. The increased alkalinity resulting from the low carbon dioxide content of the tissue fluids renders the inorganic calcium carbonate and phosphate solution unstable and accounts for the gradual deposition of these salts.

Codman,[4] Wilson,[8] Fowler [9] and others have operated on shoulders and found complete rupture of the supraspinatus tendon following such minor traumas as sudden elevation of the arm to regain balance when a person is about to fall. It seems reasonable to assume that if such minor traumas will produce a tear through the entire thickness of the tendon, it should not be an uncommon occurrence for a few fibers to be torn in the center or for an incomplete rupture to occur, with few or minor symptoms. Such a lesion would heal as any wound does and on

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5. Moschocowitz, E.: Histopathology of Calcification of the Supraspinatus Tendon as Associated with Subacromial Bursitis, Am. J. M. Sc. **150**:115-126, 1915.

6. Elmslie, R. C.: Calcareous Deposits in Supraspinatus Tendon, Brit. J. Surg. **20**:190-196, 1932.

7. Wells, H. G.: Chemical Pathology, ed. 5, Philadelphia, W. B. Saunders Company, 1925, pp. 489-496.

8. Wilson, P. D.: Complete Rupture of the Supraspinatus Tendon, J. A. M. A. **96**:433-439 (Feb. 7) 1931.

9. Fowler, E. B.: Stiff, Painful Shoulders, Exclusive of Tuberculosis and Other Infections, J. A. M. A. **101**:2106-2109 (Dec. 30) 1933.

frequent repetition of the injury would lead to areas of hyaline degeneration. Most patients who have painful calcareous deposits cannot recall having had an injury sufficient to be disabling or even inconveniencing and may well have had such a sequence of events. It seems likely, then, that deposits in the tendons about the shoulder are laid down slowly in the areas of hyaline degeneration subsequent to repair of repeated minor injuries.

In favor of this hypothesis and in accord with my findings, Carnett and Case [3b] and Moschocowitz,[5] working independently, recorded as a negative finding the absence of blood pigment in any of the many sections they examined. As seen in the roentgenograms and as reported at the time of surgical removal or at autopsy,[9a] the location of the deposit is usually in the tendon of the supraspinatus muscle near its insertion into the greater tuberosity of the humerus—the site in which rupture occurs most frequently (fig. 1). This part of the tendon lies in the groove between the tuberosities and the rounded head of the humerus and is, therefore, not the part to receive the greatest damage when squeezed between the bone and the roof of the subacromial bursa, as has been stated by those favoring the occupational theory. Also, in a series of 340 shoulders examined at autopsy, Fowler [9] described more than one third (17 of 44) of the patients with rupture of the supraspinatus tendon as having a calcareous deposit in the area of attempted healing about the defect.

One should not be misled by the term "tendinitis" as it was used by Moschocowitz [5] in rendering the first account of the histologic changes of this condition. From his descriptions of the microscopic picture it is clear that what he had in mind was mechanical inflammation and reaction to the foreign body rather than reaction to an infectious agent. One should recall, when considering the possibility of an infectious factor, that all cultures reported have been sterile. The presence of infection with the associated infiltration of inflammatory cells would increase the local metabolism and, consequently, the carbon dioxide content of the area. This would lead to an unsaturated condition of the tissue fluids—a chemical imbalance which accounts for the absorption of deposits of long standing subsequent to the reaction accompanying an acute flare-up.

A similar condition is encountered occasionally in calcification of the achilles tendon following trauma or surgical lengthening. Likewise, rider's thigh and calcification of the ligaments about the knee are conceded generally to be consequent to trauma.

9a. Keyes, E. L.: Anatomical Observations on Rupture of Supraspinatus Tendon, Ann. Surg. **97**:849-856, 1933. Keyes, E. L.: Anatomical Observations on Senile Changes in the Shoulder, J. Bone & Joint Surg. **17**:953-960, 1935. Skinner, H. A.: Anatomical Considerations Relative to Rupture of the Supraspinatus Tendon, ibid. **19**:137-151, 1937.

PATHOLOGIC PICTURE

Painter [1] rendered the first report of a case of calcareous deposit about the shoulder in 1907 but was in error as to both location and composition of the deposit, thinking it to be due to thickening of the walls of the bursa. Also, it has been suggested that this shadow-casting substance was due to the accumulation of scar tissue (Baer [10]); to fluid under pressure, a hemorrhage (Beltz [11]), and to metamorphosed fat deposits (Stern [12]). It is assumed that these investigators searched only in the bursa for the pathologic material, though Codman, in 1908, reported the surgical removal of deposits composed chiefly of calcium from beneath the bursa, in or on the supraspinatus tendon. This finding

Fig. 1.—Roentgenogram showing the usual site of calcified areas in the supraspinatus tendon. The mass was completely surrounded by tendon tissue and produced an elevation of the base of the subacromial bursa but showed no signs of local inflammation.

was soon confirmed by Wrede,[13] later by Brickner [14] and still later by others. These authors found the deposit to contain calcium and to

10. Baer, W. S.: The Operative Treatment of Subdeltoid Bursitis, Bull. Johns Hopkins Hosp. **18**:282-284, 1907.

11. Beltz, cited by Berry, J. W.: Am. J. Orthop. Surg. **14**:476-483, 1916.

12. Stern, W. G.: Metamorphosed Fat Deposits in Subdeltoid Bursitis, Surg., Gynec. & Obst. **40**:92-94, 1925.

13. Wrede, L.: Ueber Kalkablagerungen in der Umgebung des Schultergelenks und ihre Beziehungen zur Periarthritis scapulo-humeralis, Arch. f. klin. Chir. **99**: 259-279, 1912.

(Footnotes continued on next page)

be located beneath the base of the bursa, usually in relation to the tendon of the supraspinatus muscle but occasionally associated with the subscapularis and less frequently with the infraspinatus tendons.

The amorphous calcium phosphate and oxalate form a mass between the hyaline connective tissue fibers which fill the defect in the tendon (fig. 3), having no capsule or limiting membrane and being surrounded by an area of local degeneration usually the width of only a few fibers. Such calcareous deposits are supposedly never primary in the bursa (fig. 2) but lie beneath its base, in or on one of the tendons of the short rotators (fig. 4). They vary greatly in consistency, being soft

Fig. 2.—Extensive calcification of the supraspinatus tendon, which appears in the roentgenogram to be within the bursa. There were mild symptoms of thirteen years' duration, with an acute flare-up following a trauma one month previously. The floor of the subacromial bursa was smooth and glistening except for one localized reddened area (fig. 4). The deposit extended proximally within the substance of the tendon 1 inch (2.5 cm.) from its insertion. It was continuous through a sinus tract with a calcareous mass which had dissected downward to elevate the floor of the bursa for approximately 2 inches (5 cm.). Sections are shown in figures 3, 4 and 5.

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14. Brickner, W. M.: Prevalent Fallacies Concerning Subacromial Bursitis: Its Pathogenesis and Rational Operative Treatment, Am. J. M. Sc. **149**:351-364, 1915; Pain in the Arm; Subdeltoid (Subacromial) Bursitis: A Further Study of Its Clinical Types, Pathology and Treatment, J. A. M. A. **69**:1237-1243 (Oct. 13) 1917.

or hard according to the duration of the process. In the earlier stages their substance is little more than a milky fluid and resembles staphylococcus pus. With the lapse of time, the fluid is gradually absorbed; in most cases the deposits have the consistency of ointment and will exude in the form of a ribbon, as if under pressure, when the overlying tendon fibers are split. After a number of months or even years, further

Fig. 3.—Multiple calcareous deposits embedded in the hyaline connective tissue which fills the defect between the ruptured fibers of the supraspinatus tendon. Note the rounded calcified areas which have pushed the connective tissue before them as they enlarged. The roentgen appearance is shown in figure 2.

inspissation renders the deposit rather granular, dry, chalklike and finally gritty.

These deposits may occur as a number of foci (fig. 3). In 1 instance, from microscopic study of a surgical specimen, Carnett and Case [8b]

estimated that from 500 to 1,000 such areas were present. These varied from microscopic to considerable size. The larger accumulations, particularly when fluidlike, may produce an elevation under the base of the bursa resulting from localized swelling of the underlying tendon.

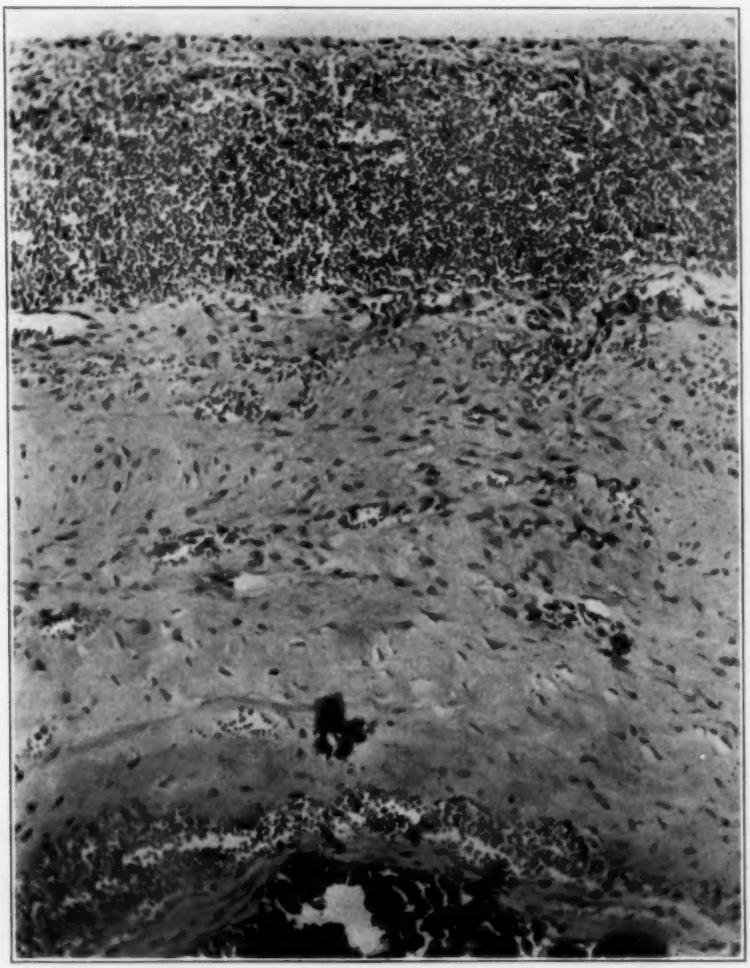

Fig. 4.—Calcified mass in the supraspinatus tendon, surrounded by hyaline connective tissue and showing a relatively normal overlying bursal floor. Note the subsynovial reaction to the mechanical irritation. The specimen was taken from the localized reddened area at the base of the bursa. The roentgen appearance is shown in figure 2.

Rarely, the deposits rupture into the bursa, producing chemical bursitis with effusion.

In reporting the microscopic observations in sections of the supraspinatus tendon from 31 shoulders, Case stated that he found cartilage in several (fig. 5) and true bone formation in 1. The local reaction varies in degree from the extensive formation of granulation tissue to the scattered infiltration of a few cells. As the lesions are noninfectious, there appears to be little attraction for polymorphonuclear leukocytes. Lymphocytes, plasma cells and large mononuclear wandering cells are the ones usually encountered (fig. 4), but in many instances foreign body giant cells may be seen. With this cellular infiltration, the fixed

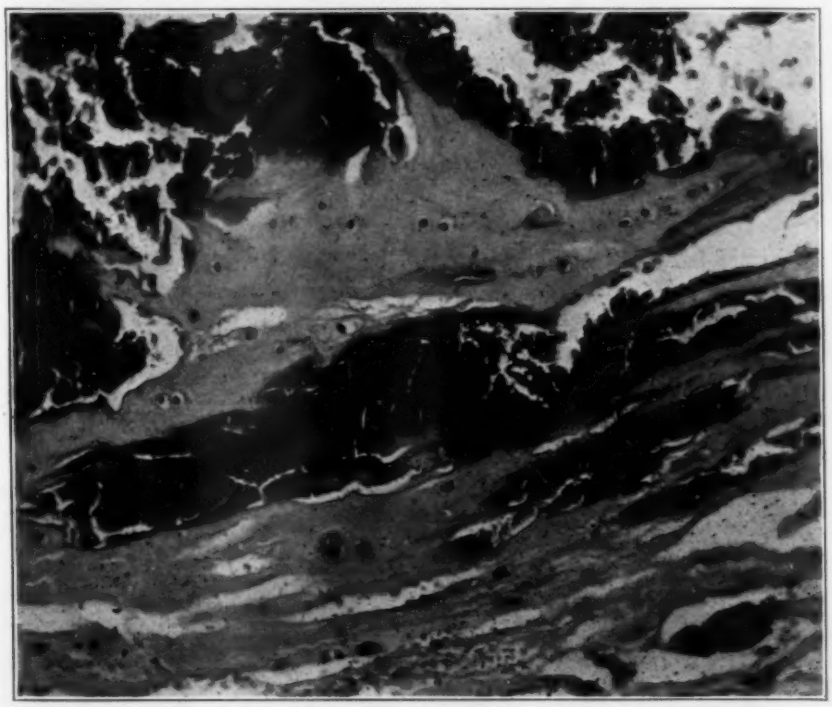

Fig. 5.—Multiple areas of calcification within the substance of the supraspinatus tendon. Note the metamorphosis of hyaline connective tissue to cartilage and the absence of a limiting membrane about the deposits, which in this area were brittle and chalklike. This is believed to represent a deposit of long standing.

tissue cells respond by proliferation, adding the fibroblast in few or greater numbers to the cellular ensemble to give a typical reaction such as would occur in the presence of any foreign substance.

The symptoms presented in general bear no relation to the size or duration of the deposit or to the amount of inflammatory reaction at the base of the subacromial bursa. It is generally believed that the deposition of the lime salts precedes by weeks, months or even years the

onset of clinical symptoms. After the deposit has formed, a mild trauma may incite acute inflammation with rapid development of the classic clinical symptoms, which probably result from the rupture of a few of the adjacent tendon fibers. The acute inflammation, with its increased blood supply and accumulation of inflammatory cells (fig. 4), "steps up" the local metabolic rate, with subsequent production of carbon dioxide in sufficient amount to lead gradually to dissolution of the calcareous deposit. The stiffness and limitation of motion accompanying this condition are due to pain and associated muscle spasm in the acute phase, but in the chronic phase they are due to contracture of the muscles and ligaments about the shoulder, which results from the prolonged voluntary fixation.

SYMPTOMS

The group of conditions responsible for disability of the shoulder joint have much in common when one is considering the onset and course of the lesion and, if not carefully studied, will seem so similar that a differential diagnosis will be impossible.

Occasionally the onset of symptoms is abrupt and viciously painful, but a detailed inquiry in most cases will elicit the history of an insidious beginning. Usually, two, six or more months before the acute onset it is noticed that there is an uncomfortable feeling associated with certain movements of the shoulder—abduction and internal rotation. Some patients find that it is uncomfortable to lie on that side and that the most comfortable position for rest is on the back, with the forearm of the troublesome side above the head; others learn to abduct the arm slightly on a pillow. Later there is a definite painful "hitch" associated with abduction of the arm through the arc from 70 to 100 degrees.

The usual sequence of events leading to an acute attack is some type of work which places an abnormal strain on the shoulder, painless at the time but recalled when symptoms develop. However, the following morning the shoulder is stiff and extremely painful on all motions. A minor trauma to the shoulder, apparently insignificant, may incite the same train of symptoms.

An acute attack may be brought about in the same way without previous subjective symptoms. Rarely, a patient cannot recall either antecedent trauma or previous symptoms of any kind.

Pain.—The pain suffered by patients with calcified deposits in the region of the shoulder should be grouped into three types, which may appear singly or in combination and may vary in degree from mild to agonizing.

It has long been recognized that many patients suffering from inflammatory conditions about the subacromial bursa feel pain only at the point of insertion of the deltoid muscle. This pain is described as sharp, cutting

or stabbing and has been compared to the pain which accompanies motion in an arthritic joint.

A greater percentage of the patients complain of a constant dull, boring or aching pain localized to the tip of the shoulder at the point of greatest tenderness. It results from accumulation of serum and inflammatory products about the deposit, which increases the pressure within the tendon and stretches the overlying synovial membrane.

There is a third type of pain associated with this and other lesions about the shoulder, which has not been overlooked but which has never been accorded its proper significance. At any time during the stage of acute or subacute symptoms the patient may suffer almost intolerable pain in the muscles of the neck, in the scapular region and occasionally down the arm as far as the finger tips. Most often it follows the distribution of the ulnar nerve, but occasionally it is encountered in the areas innervated by the median and radial nerves. It is described as shooting and burning in nature. At times there is numbness like the sensation experienced when an extremity "goes to sleep." Not infrequently there is also swelling of the involved hand. Sensory and other subjective neurologic changes are not uncommon. These findings, composing a syndrome heretofore referred to as "brachial neuritis," are identical with those encountered in the "scalenus anticus syndrome" [14a] and are believed to result from reflex spasm of the scalenus anticus muscle of the affected side.

DIAGNOSIS

From the foregoing review of the anatomy, etiology, pathology and symptomatology of these lesions of the shoulder, it can be seen that the diagnosis depends on a carefully taken history and on the physical findings. According to the symptoms, the duration of the subjective complaints and the severity of the pain with its associated muscle spasm and limitation of motion, the physical signs vary from case to case and in the same patient from day to day.

With the arm by the side, palpation will reveal a localized area of maximum tenderness below the tip of the acromion, which often coincides with an area of swelling. This may be accurately localized with reference to the bicipital groove, provided the symptoms are not too acute. There will be a painful "hitch" on abduction and again on descent of the arm. Also, on abduction the tender area will disappear beneath the tip of the acromion, a sign described by Dawbarn [15] in 1906. Abduction and rotation will be limited, but the other motions usually are essentially normal.

14a. Ochsner, A.; Gage, M., and DeBakey, M.: Scalenus Anticus (Naffziger) Syndrome, Am. J. Surg. **28**:669-695, 1935.

15. Dawbarn, R. H. M.: Subdeltoid Bursitis: A Pathognomonic Sign for Its Recognition, Boston M. & S. J. **154**:691, 1906.

However, the question of whether a calcareous deposit is present in any given case can be settled only by an adequate roentgen examination. When the calcified mass lies over the summit of the humerus (fig. 6), it may be lost in the superimposed shadow of the posterior portion of the acromion and the head of the humerus when the usual technic with the tube directly in front of the shoulder is employed. Carnett [3] has shown that deposits in this location can be thrown into relief between the acromion and the humeral head by directing the central ray slightly caudad and laterally, usually 10 to 15 degrees from the vertical in each direction.

The more usual location of these calcareous masses near the insertion of the tendons, however, presents a different problem. With this in mind, the subacromial bursa was opened to allow pieces of lead to be accurately placed between the tendon fibers. Roentgenograms were then taken with the arm in various positions. As is shown in figure 6, the importance of securing roentgenograms with the humerus in different degrees of rotation cannot be overemphasized.

Even when the position would demonstrate the deposit in silhouette, overexposure or overdevelopment will demonstrate only shadows of the more dense areas. It is therefore suggested that a "semisoft" technic be employed routinely in roentgen examination of the shoulder. This will reveal a shadow of the deposit if it is present and will also show any departures from normal in the surfaces of the adjacent bone. If stereoscopic views of the shoulder are desired, two exposures with the humerus in the neutral position and a third with the humerus in lateral rotation will prove satisfactory.

TREATMENT

In considering the treatment of patients suffering from calcareous deposits about the shoulder, one should classify the lesions into two types, the acute and the chronic. If there is a sudden onset of pain localized at the point of the shoulder and present even when the arm is at rest, the condition should be treated immediately by lavage, as advocated originally by Smith-Petersen and his associates. In the less severe attacks the patient may be kept comfortable with sedatives and an ice bag to the shoulder until the acute process has subsided. When the subacute, or chronic, stage is reached, a decision must be made to give diathermy or roentgen therapy a trial or to resort to lavage if it has not been used previously.

It must be emphasized that, although lavage may not remove completely the calcareous mass, the mechanical irritation produced by the needle usually results in infiltration of inflammatory cells, which increases the local metabolism and results in absorption of the deposit. The

Fig. 6.—*A*, roentgenogram showing lead markers within the substance of the supraspinatus tendon, one at its insertion and the other 1 inch (2.5 cm.) proximal. The arm is in the neutral position with reference to rotation. Note the superimposed shadow of the proximal marker and the acromion. The shadow of the distal marker is thrown in silhouette. *B*, roentgenogram showing the proximal lead marker in the supraspinatus tendon as in *A* and another at the insertion of the subscapularis tendon. Note the superimposed shadows of the marker in the subscapularis tendon and the head of the humerus when the arm is in the neutral position. *C*, same as *B* except the arm is in external rotation. Note the shadow of the marker at the insertion of the subscapularis tendon in this position as compared to *B*. *D*, roentgenogram taken with the shoulder in the neutral position. Note the shadow of the lateral marker, which was placed between the fibers of the infraspinatus tendon near its insertion. The proximal marker is within the supraspinatus tendon.

immediate relief often experienced by the patient probably results from the release of tension within the tendon.

In a relatively small percentage of cases the calcification and the resultant symptoms cannot be satisfactorily treated without surgical removal. This may be performed through a short exploratory incision with the region under local anesthesia.

REPORT OF A CASE

D. R., a Negress aged 37, a housewife, was referred to the Cincinnati General Hospital on Oct. 7, 1937, from the orthopedic clinic because of severe pain in the left shoulder, arm and hand.

She described the onset of a tingling and "puffed-up" sensation in her left hand about six months previously. At that time there was no discomfort in the shoulder, but after about two months she began to feel a sharp, stinging pain in the region of insertion of the deltoid muscle on elevation of the arm. Soon thereafter, lying on the affected side produced enough discomfort in the shoulder to prevent sleep. By two months before admission she had discovered a tender point at the tip of her left shoulder, where a constant dull, boring and throbbing pain was present. This was so severe at night that she slept little. It gradually grew worse, being so severe that she paid little attention to the numbness and tingling in her hand. From six weeks before admission, her left hand would regularly swell at night, the swelling disappearing during the day. She stated that she often dropped dishes or other household articles picked up with the left hand because she "could not feel them." This resulted in great anxiety; she feared that she was becoming paralyzed.

Two weeks previously, the increased pain and helplessness forced her to give up work. She sought relief by attending the medical clinic, where she was being treated for obesity. At that time her chief complaint was swelling of the left arm and hand, accompanied by numbness and tingling beginning at the left shoulder and extending down the arm to the finger tips. The general physical examination otherwise gave negative results. A diagnosis of scalenus anticus syndrome was made.

Orthopedic consultation revealed limitation of abduction and of rotation at the left shoulder by more than one half. Any movement of the joint produced pain at the point of the shoulder, where there was a localized area of tenderness just lateral to the bicipital groove. Muscular power in the left arm was weaker than in the right. The left forearm and hand were swollen, and there was diminution of sensation over the entire arm, forearm and hand to pinprick, point discrimination, heat and cold. This diminution was most pronounced over the ulnar distribution. Stereognostic sense was impaired, so that the patient could not recognize a fountain pen, a door key, a safety pin or a coin, all of which were readily identified in the right hand. There were definite tenderness and fulness in the angle between the sternocleidomastoid muscle and the clavicle on the left.

The diagnosis was changed to calcification of the left supraspinatus tendon, with spasm of the scalenus anticus muscle. Diathermy treatments were started, and roentgenograms of the shoulder and the cervical portion of the spine were taken. They did not reveal cervical rib but showed a shadow in the region of the left shoulder, just above the greater tuberosity (fig. 1). Ten days later, she had not improved with diathermy treatments and was referred to the hospital.

On admission the findings were essentially unchanged. Her chief complaint was a sensation of puffiness and numbness associated with swelling of the left hand and accompanied by sharp, burning, shooting or tingling pains down the distribution of the ulnar nerve to the finger tips. Neurologic consultants agreed with the findings and made a diagnosis of scalenus anticus syndrome. Vascular examination revealed that the left hand was 2 degrees warmer than the right and that the change in temperature with heating was greater on the left. In addition, oscillometric tracings revealed that heating resulted in the normal changes on the right but had little effect on the left.

With the region under local anesthesia, an exploratory incision was made into the subacromial bursa. When the humerus was rotated, the greater part of its floor could be inspected and was found to be entirely normal except for an elevation just lateral to the bicipital groove and just above the insertion of the supraspinatus tendon. On incision, the calcareous deposit was found to have the consistency of cottage cheese and escaped as if under pressure. Biopsy showed a deep-staining calcium deposit embedded in an area of hyaline connective tissue entirely within the substance of the tendon.

The swelling of the hand, pain in the arm and sensory changes, as well as the fulness and tenderness in the region of the left scalenus anticus muscle, disappeared within a few hours after the operation and did not return. By the fourth postoperative day, motion was greater than on admission and was painless. The patient was still asymptomatic seven months after the operation.

Comment.—This report represents 1 of 11 recent similar cases of a rather typical scalenus anticus syndrome in which the condition was entirely relieved by treatment of the calcification of the supraspinatus tendon.

SUMMARY AND CONCLUSIONS

No attempt has been made to discuss a differential diagnosis of lesions of the shoulder except in connection with cases in which calcareous deposits are present. As was shown by Codman, such deposits are most often located in the tendon of the supraspinatus muscle at the usual site of rupture, near its attachment into the greater tuberosity of the humerus. Occasionally the tendon of the infraspinatus or the subscapularis muscle is involved. The calcium salts are thought to be laid down slowly over a period of months or even years in the hyaline connective tissue subsequent to repair of repeated minor traumas. The masses are asymptomatic until they are large enough to produce mechanical disturbances or until a minor trauma tears a few of the adjacent tendon fibers and produces mechanical irritation with the accumulation of serum and inflammatory cells to activate the process.

A case is presented to illustrate the types of pain encountered. Pain in the region of the insertion of the deltoid muscle is thought to be referred from the subacromial bursa. The increased pressure within the tendon and the stretching of the overlying synovial membrane which lines the base of the bursa seem to account for the occurrence of constant dull, boring or aching pain localized to the point of the shoul-

der. The third type of pain encountered in this condition is really the result of a complication. It consists of pain throughout the distribution of the brachial plexus. The most severe symptom is a burning, shooting or tingling sensation down the arm, most often in the distribution of the ulnar nerve but also encountered in the areas innervated by the median and radial nerves. It is often associated with swelling of the involved hand. Oscillometric tracings may show some decrease in the vascular pulsations on the affected side. Sensory and other subjective neurologic changes are not uncommon. The entire picture is that presented by the scalenus anticus syndrome and is thought to result from reflex spasm of the scalenus anticus muscle of the affected side.

The diagnosis depends on a carefully taken history and on the physical findings, which, however, differ little from those associated with other painful conditions of the shoulder. Roentgen examination should consist of the taking of anteroposterior views of the shoulder with the humerus in the neutral position and in lateral rotation, a "semisoft" technic being advisable.

Routine treatment is considered radical. The acute condition should be treated immediately by lavage. In the subacute or chronic stages, a decision must be made to give diathermy a trial or to resort to lavage if it has not been used previously. In a small percentage of cases the condition cannot be satisfactorily treated except by surgical removal of the deposit.

The cases mentioned in this report were studied under the supervision of Dr. J. A. Freiberg.

ACUTE PANCREATIC NECROSIS AND ACUTE INTERSTITIAL PANCREATITIS

TREATMENT WITHOUT OPERATION: A CLINICAL STUDY OF TEN CASES

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Acute pancreatic necrosis is a term usually applied to a serious, often fatal, disease of the pancreas which is due to autodigestion of the gland, presumably by activation within the ducts of trypsinogen to trypsin, the latter being a powerful proteolytic ferment. The classic observations of Fitz,[1] based on necropsy study, have long been the basis for physicians' knowledge of this remarkable condition. The mortality is very high even if operation is carried out, although it has been generally agreed that the disease is primarily surgical and that surgical intervention offers the greatest hope of survival.

For the past decade or so a growing experience has shown that many patients with acute pancreatitis are cured without operation and that many are cured even if nothing more than exploration is done at operation. According to this point of view, acute pancreatitis tends to become less and less a surgical disease, in the sense that operation is not indicated and may even prove deleterious. Although the pendulum is thus swinging from operative to nonoperative therapy, there is good reason to suspect that the true state of affairs lies somewhere between the two extremes. This has been suggested by evidence indicating that there are two types of acute pancreatitis, one a self-limited inflammation or obstruction which subsides spontaneously and has been designated as acute interstitial pancreatitis and the other, which is the serious, often fatal, type, being most appropriately described as acute pancreatic necrosis.

The present clinical study is based on 5 cases of each type, in all of which the condition was primarily treated without operation. The differences between the two groups were so striking that it seemed worth while to summarize them with the view of suggesting a form of

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1. Fitz, R. H.: Acute Pancreatitis: A Consideration of Pancreatic Haemorrhagic, Suppurative, and Gangrenous Pancreatitis, and of Disseminated Fat-Necrosis, Boston M. & S. J. **120**:181-229, 1889.

therapeutic approach that might tend to lower the mortality, which in the cases of acute pancreatic necrosis was 100 per cent.

In the first 5 cases reported, the diagnosis was acute interstitial pancreatitis; in the second group, acute pancreatic necrosis.

REPORT OF CASES

CASE 1.—A 42 year old Armenian man entered the hospital on Dec. 14, 1937, complaining of severe epigastric pain with a fairly sudden onset forty-eight hours prior to entry. The pain was constant and did not radiate. The patient vomited once and had several watery stools after self medication with "salts." There was no history of jaundice. He had had one similar attack six months previously, milder than the present one.

Physical Examination.—The patient was well nourished, swarthy and somewhat obese. He appeared rather acutely ill. There was no visible evidence of jaundice. The abdomen moved freely with respiration, and there were no visible masses. Palpation of the abdomen showed tenderness and voluntary muscle guard over the epigastrium and the right upper quadrant. There was no rigidity. No masses were felt.

Laboratory Examination.[2]—The urine gave a 1 plus reaction for albumin but contained no sugar. The leukocyte count was 14,000 per cubic millimeter. The Kahn reaction was negative. The value for sugar was 114 mg. and that for nonprotein nitrogen 24 mg. per hundred cubic centimeters of blood (both normal). The value for blood amylase on December 14 at 6 p. m. was 200; at 11 p. m. the same day it was 250. On December 15 it was 250. On December 16 and 17 it returned to normal, i. e., 33 and 30, respectively. The icterus index remained normal.

A roentgenogram taken on December 14 disclosed no free air under the diaphragm. One week after the patient's admission a cholecystogram taken after intravenous injection of soluble iodophthalein U. S. P. revealed a pathologic gallbladder (no shadow).

Course.—The patient improved steadily with a diet high in carbohydrates. Two days after admission he was free from symptoms. Cholecystectomy was advised, but he refused the operation and left the hospital.

Two months later he was readmitted, with complaints similar to those noted at the time of his first entry, plus radiation of the epigastric pain to the back. The attack had commenced eighteen hours prior to entry. The value for blood diastase, determined twenty-four hours later, was 67, and forty-eight hours later it was 33, the latter value being normal. Three days after admission, all acute symptoms having subsided, an abdominal exploration was performed, and the pancreas was said to be acutely injected but fairly normal to palpation. The gallbladder was fibrotic and adherent. No stones were found in the common bile duct, which was not dilated. A cholecystectomy was performed, and the common duct was drained by a T tube. A biopsy of pancreatic tissue taken at the time

2. Chemical methods were standard except for that used to determine the amylase content of the blood, which was the procedure recently described by M. Somogyi (Studies on Blood Diastase, Proc. Soc. Exper. Biol. & Med. **29**:1126-1128 [June] 1932; Blood Diastase as Indicator of Liver Function, ibid. **32**:538-540 [Dec.] 1934).

of operation revealed marked fibrosis of the interacinar framework and some infiltration with lymphocytes. There was no necrosis or suppuration. The pathologic diagnosis was chronic pancreatitis. The liver showed considerable fatty degeneration; the gallbladder revealed only slight change.

CASE 2.—A white woman aged 60 entered the hospital on April 30, 1937, complaining of severe pain in the epigastrium and both upper quadrants of the abdomen, which radiated to the back along both costal margins. The onset had taken place about six hours before entry, and the patient had vomited several times. She described similar previous attacks, which were milder, though sequentially increasing in severity.

Physical Examination.—The patient was very obese and acutely ill. Tenderness was elicited across the entire upper part of the abdomen. There were no palpable masses and no muscle guard. There was no jaundice.

Laboratory Examination.—The urine showed no albumin or sugar. The leukocyte count was 8,640 and the erythrocyte count 3,800,000 per cubic millimeter of blood. The Kahn reaction was negative. The value for blood sugar was 122 mg. and that for nonprotein nitrogen was 21 mg. per hundred cubic centimeters. The value for blood amylase on May 1 was 333 in the morning and 225 in the afternoon. On the two successive days the values had fallen to 182 and 28, respectively.

Course.—Three days after admission all symptoms had subsided and the patient was discharged.

CASE 3.—A white man aged 65 entered the hospital on June 29, 1937, complaining of severe epigastric pain which had begun six hours prior to entry and was steadily becoming worse. The patient was nauseated and vomited several times. He had had several similar attacks before this, one accompanied by jaundice in 1935. The patient was being treated by a private physician for "ulcer of the stomach."

Physical Examination.—The patient was well nourished. He was in acute distress. There was visible jaundice. Palpation over the epigastrium was painful. There was some tenderness in both subcostal areas, particularly on the left. There was no rigidity, and no masses could be outlined.

Laboratory Examination.—The diastase content of the urine was 4,000; otherwise the urine was normal. The value for blood sugar was 158 mg. and that for nonprotein nitrogen was 27 mg. per hundred cubic centimeters. The leukocyte count was 7,100 per cubic millimeter. The Kahn reaction was negative. The icterus index was 50. The blood amylase on the morning of June 29 was 400 and on the afternoon of the same day was 500. On June 30 and July 2 the figures had dropped to 165 and 20, respectively.

Roentgen examination for free air under the diaphragm gave negative results at the time of the patient's admission, and on July 9 a cholecystogram taken after intravenous injection of soluble iodophthalein U. S. P. was normal.

Course.—Three days after the patient's admission the symptoms had subsided, and six days later he was discharged.

CASE 4.—A white man aged 69 entered the hospital on Dec. 2, 1935, complaining of severe pain in the midline just above the umbilicus. The onset had taken place one week previously and had been accompanied with jaundice. There was no history of radiation of pain. The condition had become progressively worse

until the patient sought hospitalization. He was nauseated and vomited frequently. There was a history of previous attacks, milder than the present episode.

Physical Examination.—The patient was well developed and well nourished. There was an icteric tinge to the skin. The abdomen was obese and tender to palpation over the epigastrium and the right upper quadrant of the abdomen. There was no rigidity, and the examiner described a "vague mass in the right upper quadrant."

Laboratory Examination.—The urine showed a trace of albumin and gave a strongly positive reaction for bile. The leukocyte count was 24,200 and the erythrocyte count 5,360,000 per cubic millimeter. The Kahn reaction was negative. The value for sugar was 99 mg. and that for nonprotein nitrogen was 35 mg. per hundred cubic centimeters of blood. The amylase content of the blood on December 4 (thirty-six hours after admission) was 110, and on the next day it was 25. The icterus index was 90.

Past History and Course.—Fifteen months prior to this entry the patient had been hospitalized because of a similar attack. His condition was diagnosed as cholelithiasis, and roentgenograms of the gallbladder taken after intravenous injection of soluble iodophthalein U. S. P. (Sept. 27, 1934) showed pathologic functioning. The patient was discharged five days after entry.

CASE 5.—A white woman aged 27 entered the hospital on Aug. 7, 1936, complaining of extreme pain in the epigastrium, nausea and vomiting which began about thirty-six hours prior to entry. The pain was persistent and stabbing, frequently radiating to the interscapular region. No history of jaundice was obtained. The patient had had two previous attacks within four months previous to hospitalization.

Physical Examination.—The patient was moderately obese. She was in acute distress. Palpation revealed fulness in the epigastrium with tenderness confined to the region of the pancreas and a typical "Head" zone of cutaneous hyperesthesia. There was voluntary muscle guard over the entire upper part of the abdomen, but there was no rigidity.

Laboratory Examination.—The urine gave a 2 plus reaction for albumin but was otherwise normal. The leukocyte count was 20,400 per cubic millimeter on the patient's admission and 8,900 six days later. The Kahn reaction was negative. The value for blood sugar was 155 and that for nonprotein nitrogen was 24 mg. per hundred cubic centimeters. The amylase content of the blood on the day of admission was 330; on the following seven successive days it fell to 160, 115, 15, 18, 20, 28 and 20. The icterus index was normal.

Roentgen examination of the gallbladder after intravenous injection of soluble iodophthalein U. S. P. revealed it to be pathologically functioning (no shadow).

Course.—The patient's symptoms had subsided three days after admission, and on August 26 a cholecystectomy was performed. There were many stones in the gallbladder. The common duct showed no changes; the pancreas was not examined. The patient died on the fifth postoperative day. Permission for autopsy was refused.

CASE 6.—A white man aged 61 entered the hospital on Dec. 11, 1935, complaining of severe epigastric pain of five hours' duration. The pain was persistent. It did not radiate to the back but was accompanied by nausea and vomiting. There was no history of a similar attack, and the patient stated that he had never had jaundice. There was a history of cardiac disease and digitalization.

The patient was not in acute distress. The heart was enlarged; the sounds were irregular. The abdomen moved with respiration and was moderately distended. The liver was enlarged. There was tenderness in the epigastrium and to a lesser degree in both upper quadrants of the abdomen. Voluntary muscle guard over the entire upper part of the abdomen was encountered, but there was no rigidity.

Laboratory Examination.—The urine was essentially normal. The leukocyte count was 7,400 per cubic millimeter. The Kahn reaction was negative. The value for blood sugar was 90 mg. and that for nonprotein nitrogen 38 mg. per hundred cubic centimeters. The amylase content of the blood on December 12 was 200 both in the morning and in the afternoon. The following day it fell to 166. December 16, 18, 21 and 23 the readings were 10, 30, 40 and 28, respectively. The icterus index was 75.

A roentgenogram taken on admission showed no free air under the diaphragm.

Course.—The patient became progressively worse, the symptoms being localized in the epigastrium and the right upper abdominal quadrant. The white blood cell count was elevated to 43,000 on December 23, and on the evening of the same day, twelve days after admission, the patient died.

Postmortem Examination.—The chief cause of death was acute necrotic pancreatitis. There was a large amount of free purulent fluid within the abdominal cavity, and the peritoneum and mesentery were studded with chalky areas of necrosis, with numerous pockets of pus and débris between adherent loops of intestine. The omentum was thick and friable. The liver was was not enlarged but gave microscopic evidence of chronic hepatitis. The gallbladder was filled with small stones and purulent bile, and the cystic and common ducts were dilated, the latter containing about a dozen small faceted stones. The pancreas was swollen, necrotic and black. The pancreatic duct was patent and bile stained. Microscopically whole sections of the pancreas were necrotic, with some round cell infiltration into the bordering tissues.

CASE 7.—A white woman aged 81 entered the hospital on March 19, 1937, complaining of generalized abdominal pain, nausea and vomiting of three days' duration. The pain was most marked in the epigastrium, and the patient stated that it did not radiate. There was no history of similar previous attacks.

Physical Examination.—The patient was in acute distress. She was cyanotic and had a grayish pallor. The abdomen moved with respiration and was not rigid. There were no borborygmi. The upper part of the abdomen was tender and exhibited voluntary muscle guard.

Laboratory Examination.—The urine gave a 1 plus reaction for albumin. The leukocyte count was 13,800 per cubic millimeter. The Kahn reaction was negative. The value for blood amylase was not determined.

Course.—The patient became progressively worse and died on the day after admission. The diagnosis was partial intestinal obstruction.

Postmortem Examination.—The main cause of death was acute pancreatic necrosis. Grossly, the distal two thirds of the pancreas was entirely necrotic and hemorrhagic. The peritoneal cavity contained several hundred cubic centimeters of bloody fluid. The gallbladder was moderately distended and contained about fifteen small stones. The common bile duct was slightly dilated but contained no stones. The main pancreatic duct was patent and contained no calculi or bile. Microscopically, a section through the head of the pancreas showed little change

except some round cell infiltration into the peripancreatic fat. Sections from the body and tail of the pancreas revealed complete destruction of pancreatic tissue.

CASE 8.—A white woman aged 33 entered the hospital on Nov. 1, 1937, complaining of abdominal pain, nausea, vomiting and chills. The patient had been feeling "under par" for three weeks but noticed the severe abdominal pain only four days prior to entry. She also complained of repeated chills and of burning on urination.

Physical Examination.—The patient was moderately obese. She was in shock. The pulse was rapid and thready. The skin was cold, and the temperature was subnormal. There were tenderness and voluntary muscle guard over the entire abdomen, most marked in the upper quadrants. There was marked tenderness in both costovertebral angles. The chest was essentially normal.

Laboratory Examination.—The urine gave a 3 plus reaction for albumin. The leukocyte count was 26,350 per cubic millimeter. The Kahn reaction was negative. The value for sugar was 252 mg. and that for nonprotein nitrogen 42 mg. per hundred cubic centimeters of blood. The value for blood amylase was not determined.

Course.—Two days after admission the patient had a sudden fairly severe hemoptysis, and the entire chest revealed moist, coarse rales on auscultation. Roentgenograms taken with an emergency portable apparatus at the bedside revealed blotchy areas of consolidation over both lung fields, suggestive of extensive bronchopneumonia or acute pulmonary edema. The patient died three days after admission, and a diagnosis of tuberculous pneumonia was made.

Postmortem Examination.—The prime cause of death was acute pancreatic necrosis. The lungs showed no pneumonic process but were edematous. The peritoneal cavity contained about 250 cc. of straw-colored fluid. The gallbladder and bile ducts appeared normal. Grossly, the entire pancreas contained fatty necrotic areas and multiple hemorrhagic spots. The ducts showed no calculi or evidence of dilatation. Microscopically, all sections showed extensive necrosis; even the supportive structures in some areas were unrecognizable.

CASE 9.—A white man aged 34 entered the hospital on June 10, 1936, and was sent to the ward for patients with alcoholism because of a strong alcoholic odor to his breath. He gave a history of heavy imbibing of alcoholic beverages during the past ten days. However, two days prior to entry he noticed pain and distention in the upper part of the abdomen and was thereafter unable to retain either food or water. There was no history of radiation of pain or of previous attacks. His bowels had been moving rather loosely.

Physical Examination.—The patient was moderately obese. He was sitting up in bed. He was moderately dyspneic. He was conscious and rational but rather apprehensive. The abdomen was distended but showed no rigidity. There was considerable tenderness over the right upper quadrant, and the edge of the liver was palpated slightly below the right costal margin. Voluntary muscle guard was present over the upper part of the abdomen. No abnormal masses were palpated. Shifting dulness and a fluid wave were demonstrated.

Laboratory Examination.—The urine gave a 4 plus reaction for albumin and for urobilinogen. The leukocyte count was 4,600 and the erythrocyte count 3,920,000 per cubic millimeter. The Kahn reaction was negative. The value for sugar was 149 mg. and that for nonprotein nitrogen was 23 mg. per hundred cubic centimeters of blood. The icterus index was 75. The value for blood amylase was not determined.

Course.—The patient became progressively worse. On November 13, three days after admission, he went into shock and died. The diagnosis was alcoholic enteritis.

Postmortem Examination.—The cause of death was acute pancreatic necrosis. The peritoneal cavity contained about 500 cc. of bloody fluid, and there were numerous chalky white areas of fat necrosis on the surface of the greater omentum. The liver was enlarged and showed evidence of fatty degeneration. The gallbladder was adherent to the omentum and on pressure expelled bile through the ampulla of Vater. In the region of the pancreas there was a large, dark red friable mass surrounded by omentum. There was considerable fat necrosis about the pancreas. The main duct was patent and contained no stones. Microscopic section showed the tail to be entirely necrotic; the body and head showed fairly normal acinar tissue with fat necrosis in the interacinar fibrous tissue.

CASE 10.—A white man aged 42 entered the hospital on June 16, 1936, complaining of pain in the epigastrium, nausea and vomiting. At the onset, which had taken place two days prior to entry, the pain was not severe, but four hours later it became alarming and a physician was called in. He described the pain as commencing in the epigastrium, radiating to the right and finally including the entire abdomen. The patient vomited about twelve times. No history of any similar previous attack was reported. There was a vague history of jaundice.

Physical Examination.—The patient was obese. He was in acute distress and in mild shock. The abdomen was distended but not rigid. Tenderness was elicited in the epigastrium and in the right upper quadrant. A large mass was palpated across the epigastrium.

Laboratory Examination.—The urine on admission was essentially normal. The leukocyte and erythrocyte counts were 16,350 and 5,050,000 per cubic millimeter, respectively. The Kahn reaction was negative. The value for blood sugar was 213 mg. and that for nonprotein nitrogen was 63 mg. per hundred cubic centimeters. The value for blood amylase was 20 and remained normal or low throughout the disease. The icterus index was 63. The organism in the culture of material taken from the abdominal cavity at the time of operation was reported as Staphylococcus albus.

Course.—The patient slowly improved under treatment with a conservative regimen plus blood transfusions. On July 8 the following notes were made by the resident: "Patient still presents the picture of chronic illness. Has a large mass in the upper part of the abdomen. Believe this is a condition which originated with disease of the gallbladder and went on to cause acute pancreatitis. . . . Believe the patient had a localized peritonitis or abscess in the lesser peritoneal cavity, which then extended to the right to give a subphrenic abscess. . . . This seems to be confirmed by a roentgenogram of the chest which shows a high elevation of the diaphragm on the right. Two days previously the patient began to complain of severe pain in the left lower part of the chest. Examination revealed a loud, rough friction rub."

On July 12, the patient had been in the hospital over three weeks, an exploratory laparotomy was performed; this revealed a large, fairly well walled-off abscess which extended beneath the liver and also communicated with a similar process over the pancreas. On July 15, after a rather stormy postoperative course, the patient died.

Postmortem Examination.—There was a large abscess cavity beneath the liver, which was well walled off from the greater peritoneal cavity. The abscess extended

into the lesser peritoneal space and retroperitoneally, where it enclosed the pancreas. It contained a mass of necrotic friable material the structure of which resembled that of the pancreas. The liver was normal in size and shape, and there was no evidence of subdiaphragmatic abscesses. The common bile duct and the gallbladder showed normal patency and contained no stones. Microscopically the pancreas revealed large areas of edematous necrotic tissue. There were also large areas of uninvolved pancreas, and in these regions considerable fibrosis was observed.

COMMENT

The data presented in this report are really self explanatory. Much of the significant material has been summarized in the accompanying chart and table. Further presentation of the findings will be correlated with a brief discussion of the problem as well as a review of the recent literature under appropriate headings.

Classification.—Until 1933 the term acute pancreatitis meant in general but one disease, namely, the acute fulminating type of pancreatic necrosis. At that time, Elman [3] presented evidence, supported by careful historical and clinical analysis, pointing to a disease entity which he termed acute interstitial pancreatitis. He felt able "to justify the conclusion that they were dealing with a type of acute pancreatitis with edema, swelling or induration which was distinct from the usual cases of acute pancreatitis in showing no evidence of gland necrosis, hemorrhage or suppuration." His description of the macroscopic, microscopic and clinical pictures probably warrants a separation of this type of acute pancreatitis into a class by itself. Although certain authors have claimed otherwise, Elman stated that this entity is "not merely an early stage in the development of frank pancreatic necrosis." Even if Elman's interpretation is wrong, there is good reason to believe that this pathologic cycle, which subsides prematurely, produces an independent clinical picture. The first group of cases described in this paper were of this type.

The second group of cases described here is differentiated from the first by a much more severe reaction, with necrosis, hemorrhage and even suppuration of the pancreas. This condition has found considerable space in the surgical literature and as a result has become rather familiar, although acute pancreatic necrosis is not a common malady. Another term used almost interchangeably with acute pancreatic necrosis is acute hemorrhagic pancreatitis, for necrosis and hemorrhage frequently go hand in hand.

Symptomatology.—Fitz,[1] in 1889, made the statement:

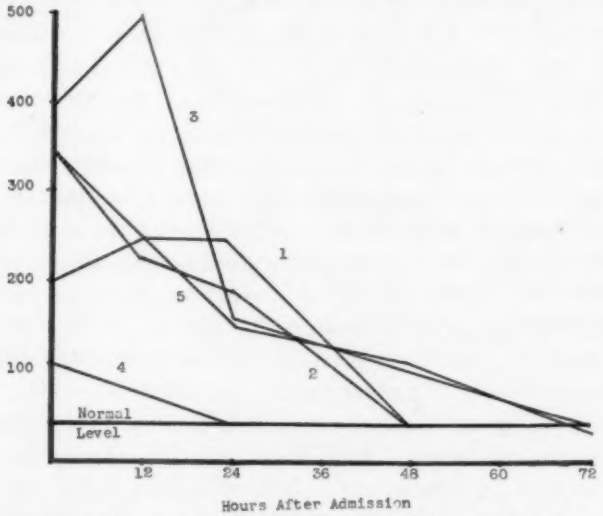
Acute pancreatitis is to be suspected when a previously healthy person, or sufferer from occasional attacks of indigestion, is suddenly seized with violent

3. Elman, R.: Acute Interstitial Pancreatitis, Surg., Gynec. & Obst. **57**:291-309 (Sept.) 1933.

pain in the epigastrium followed by vomiting and collapse, and, in the course of twenty-four hours, by a circumscribed epigastric swelling, tympanitic or resistant, with a slight rise in temperature.

He was, of course, referring to the condition called acute pancreatic necrosis.

The symptoms of acute interstitial pancreatitis, although similar to those of acute pancreatic necrosis, are not fulminating and do not produce shock and circulatory collapse but have a tendency to subside in a few hours or days. A point of interest in this series is the history of previous, though milder, attacks in the cases of interstitial pancreatitis.



Curves representing the amylase content of the blood in 5 cases of acute interstitial pancreatitis. The patient in case 4 entered the hospital with a subsiding attack which had begun one week previously. The relatively low curve is probably due to this fact.

Summary of Important Observations in Two Groups of Cases of Acute Pancreatitis Treated Without Operation

	Acute Interstitial Pancreatitis					Acute Pancreatic Necrosis				
Case	1	2	3	4	5	6	7	8	9	10
Age	42	60	65	69	27	61	81	33	34	42
Sex	M	F	M	M	F	M	F	F	M	M
Previous attacks	Yes	Yes	Yes	Yes	Yes	No	No	?	No	No
Epigastric pain and vomiting	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Prostration (severe)	No	No	No	No	No	Yes	Yes	Yes	Yes	Yes
High value for blood amylase	Yes	Yes	Yes	Yes	Yes	Yes	?	?	?	No
Leukocytosis	Yes	No	No	Yes	Yes	No	Yes	Yes	No	Yes
Blood sugar, mg./100 cc.	114	122	158	158	155	90	?	252	149	213
Jaundice	No	No	Yes	Yes	No	Yes	?	?	Yes	Yes
Termination	Recovery	Recovery	Recovery	Recovery	Recovery	Death	Death	Death	Death	Death

The outstanding and constant symptom of acute pancreatitis, regardless of type, is the severity of the pain. One has only to review a few authentic case histories and observe the frequency of a misdiagnosis of perforated peptic ulcer to realize the type and intensity of pain. The location is not constant, and this has been a source of confusion. Although the patient usually points to the epigastrium as the site of disturbance, he may also direct attention to either the right or the left upper quadrant of the abdomen, or he may complain of generalized abdominal pain. Radiation of pain is also described, either straight through from the epigastrium to the interscapular region or to the small of the back. Nausea and repeated vomiting are the rule.

Careful palpation will frequently disclose an area of tenderness in the epigastrium and the left side of the hypochondrium, over the region of the pancreas. Elman stated: "In a few cases I was able to outline a Head-zone of skin hyperesthesia along the left costal margin."[4] Often, however, the tenderness may be diffuse over the entire upper part of the abdomen, which may sometimes be distended and tympanitic to percussion, with the "silence" of peritonitis. This finding is easily explained by stimulation of the celiac and superior mesenteric ganglions and plexuses, which lie close to the pancreas. Distention probably explains the occasional misdiagnosis of intestinal obstruction.

In cases of acute pancreatic necrosis there is generally definite evidence of collapse, frequently accompanied by cyanosis. This condition gives all of the picture produced by shock, such as a fall in blood pressure, rapid, weak and thready pulse, cold and clammy skin and apprehension on the part of the patient. De Klimkó[5] described his cases in three groups, according to the clinical picture: The first group includes those with a sudden onset and rapid progress with death in a short time, autopsy showing extensive fat necrosis and pancreatic necrosis. In the second group the symptoms are less severe and tend to subside. The last group comprises the cases of mild involvement. Obviously, in a great portion of the second and third groups the disease would fit in with the type described in this report as acute interstitial pancreatitis.

Many patients show frank jaundice; others have an icteric scleral tinge. This is probably explained by the course of the common duct through the head of the pancreas, where it may readily be compressed in a case of pancreatic edema or tumor. There is usually mild leukocytosis, the increased leukocyte count being scarcely significant the first day but rising to 15,000 or 20,000 on the second day. The erythrocytes

4. Elman, R.: The Diagnosis and Treatment of Acute Pancreatitis, Am. J. Digest. Dis. & Nutrition **4**:732-736 (Jan.) 1938.

5. de Klimkó, D.: Surgical Treatment of Acute Pancreatitis, Surg., Gynec. & Obst. **63**:89-95 (July) 1936.

show little change. The urine may occasionally contain sugar during the acute phase of the attack and at times may contain urobilinogen.

The cornerstone in the diagnosis of acute pancreatitis lies in the determination of the value for diastase (amylase) in the blood. Much has been written on the biochemistry of blood diastase, and much more remains to be made clear. There is not complete agreement as to its source or function (Clasen, Johnstone and Orr [6]). Elman, Arneson and Graham [7] reported observations in the human being which led them to believe that a low value for blood amylase meant destruction of the acinar tissues of the pancreas, whereas increased amylase in the blood resulted from obstruction of the ducts. This would explain how early in the disease there may be a high value for blood amylase, followed later, if there is considerable pancreatic necrosis, by a low value. In interstitial pancreatitis also a high level occurs early, but the progressive fall, usually in a day or two, has a different significance, being closely correlated with the subsiding symptoms. Somogyi [8] has correlated low values for blood diastase with severe hepatic injury.

The sugar tolerance curve has been used by de Klimkó [5] as an aid in diagnosis. He asserted that for patients with subsiding acute pancreatitis the curve shows higher values during the first thirty minutes and does not fall to the normal for some time. He called attention also to an elevation of the serum lipase in this condition, which he attributed to the fat necrosis associated with acute pancreatitis. It is of interest to note the elevation of the cholesterol content of the blood in many of the cases of acute pancreatitis.

Differential Diagnosis.—Acute cholecystitis or biliary colic, perforated duodenal or gastric ulcers, intestinal obstruction and acute coronary thrombosis are the diseases which usually present a problem in the differential diagnosis of acute pancreatitis. The diagnostic error lies not so much in the lack of a thorough work-up as in the failure to consider acute pancreatitis as a possibility in every case of disease of the upper part of the abdomen. In case of doubt in diagnosis the diastase content of the blood should always be determined. In a series of 18 cases of acute pancreatitis, in all of which the characteristic curves for blood amylase were obtained, Elman [3] showed that in 9, or 50 per cent, the diagnosis on admission was biliary colic or acute cholecystitis. In cases of the interstitial type of acute pancreatitis, as in cases of chronic

6. Clasen, A. C.; Johnstone, P. N., and Orr, T. G.: Blood Amylase in Experimental Pancreatitis, Surg., Gynec. & Obst. **59**:756-761 (Nov.) 1934.

7. Elman, R.; Arneson, N., and Graham, E. A.: Value of Blood Amylase Estimations in Diagnosis of Pancreatic Disease: Clinical Study, Arch. Surg. **19**: 943-967 (Dec., pt. 1) 1929.

8. Somogyi, cited in footnote 2.

cholecystitis, there is frequently a history of previous attacks with absence of symptoms during the interval. Every case of supposed biliary colic should be studied with pancreatitis in mind; there will frequently be a high value for blood diastase. A perforated peptic ulcer will give in most cases a typical and diagnostic clinical picture. The suddenness of onset and the location and character of the pain may be reproduced by acute pancreatitis. However, a past history of ulcer, the presence of abdominal rigidity and especially roentgenograms showing air under the diaphragm are all diagnostic aids. It must be kept in mind that an ulcer on the posterior surface of the stomach may rupture into the lesser peritoneal cavity and evoke an elevation of the amylase content of the blood as a result of direct irritation of the pancreas as by the contents of the stomach (Probstein, Gray and Wheeler [9]).

Acute intestinal obstruction when high and accompanied (as it is) by frequent vomiting will sometimes rather closely simulate acute pancreatitis. However, the peristaltic rushes, the roentgen pictures, the fluctuating character of the pain and the presence of a cause should dispel any confusion.

Etiology.—The older literature has so frequently been cited that it will not be detailed here. A recent review is that of Robins.[10] This author, as have others, notably Opie,[11] emphasized the entrance of bile into the pancreatic ducts as the most frequent cause. In 1921, Archibald [12] discussed this phase of the disease and stated the conviction that the regurgitation of bile into the pancreatic system is the usual causative factor. Three years later, Eggers [13] advanced his hypothesis that the lesion in acute pancreatitis is due to the release of pancreatic juices into the adjoining tissues, and he added that infection in itself has little to contribute to the pathologic picture. In 1933, Finney [14] summed up the matter by stating that there is activation within the gland of pancreatic ferments by a reflux of bile, causing a chemical necrosis in which infection plays a secondary part. Important in this connection is the experimental transplantation of living pancreatic tissue into a window of

9. Probstein, J. G.; Gray, S., and Wheeler, P. A.: Blood Diastase in Acutely Perforating Peptic Ulcers, Proc. Soc. Exper. Biol. & Med. **37**:613-615 (Jan.) 1938.

10. Robins, C.: Bile Tract and Acute Pancreatitis, Ann. Surg. **103**:875-885 (June) 1936.

11. Opie, E. L.: Disease of the Pancreas: Its Cause and Nature, ed. 2, Philadelphia, J. B. Lippincott Company, 1910, pp. 15 and 200.

12. Archibald, E.: Further Data Concerning the Experimental Production of Pancreatitis, Ann. Surg. **74**:426-433 (Oct.) 1921.

13. Eggers, C.: Acute Pancreatitis, Ann. Surg. **80**:193-209 (Aug.) 1924.

14. Finney, J. M.: Pancreatic Emergencies, Ann. Surg. **98**:750-759 (Oct.) 1933.

the duodenum, as accomplished by Dragstedt.[15] This produced no digestion, whereas when the pancreas was implanted into the gallbladder there was necrosis of the pancreatic tissues exposed to the bile. There are two possibilities in the hydrodynamics of the retrojection of bile into the pancreatic duct system: first, mechanical obstruction due to an impacted stone and, second, spasm of the sphincter of Oddi. The latter is anatomically possible in at least 20 per cent of all adults, according to pathologic and anatomic studies. Autopsies and examination of the common duct during operative procedures have shown sufficient evidence to place this factor on a definite etiologic basis. The benefit of cholecystectomy as a therapeutic measure is said to be in the fact that after this procedure there is dilatation of the common duct and of the sphincter of Oddi. That there is a close relation between disorders of the biliary system and acute pancreatitis is a matter of agreement in the minds of most authors who have discussed this subject. Statistics[16] reveal the rather convincing fact that as many as 70 per cent of patients with acute pancreatitis have associated disease of the gallbladder.

It has recently been demonstrated that in certain cases the administration of amyl nitrite relieves the severe pain of acute pancreatitis, probably by relaxing the sphincter of Oddi. Amadon[17] reported a case of acute pancreatitis in which during operation agenesis of the gallbladder was revealed. This observation was regarded by the author as significant in that it indicated a disturbance in the pressure balance normally present in the biliary system, which permitted a regurgitation of bile into the pancreatic duct. Pavel,[18] in discussing jaundice caused by spasm of the sphincter of Oddi, stated that it is difficult to appreciate the exact nature and location of the lesions giving rise to this reflex spasm. He added that inflammation in the gallbladder, common bile duct, pancreas and duodenum may initiate a spastic condition of the sphincter.

A contrasting view of the etiology of acute pancreatic necrosis is that of Rich,[19] who minimized the role of bile and presented evidence that metaplasia of the epithelium of the ducts may play an important role.

Death from acute pancreatitis is due to toxemia resulting from absorption of necrotic glandular elements. The toxicity of these sub-

15. Dragstedt, L. R.; Haymond, H. E., and Ellis, J. C.: Pathogenesis of Acute Pancreatitis (Acute Pancreatic Necrosis), Arch. Surg. **28**:232-291 (Feb.) 1934.

16. Schmieden, V., and Sebening, W.: Chirurgie des Pankreas, Arch. f. klin. Chir. **148**:319, 1927.

17. Amadon, P.: Agenesis of the Gall Bladder Associated with Pancreatitis, Am. J. Surg. **19**:263-267 (Feb.) 1933.

18. Pavel, I.: Jaundice Caused by Functional Obstruction, J. A. M. A. **110**: 566-569 (Feb. 19) 1938.

19. Rich, A. R., and Duff, G. L.: Experimental and Pathological Studies on the Pathogenesis of Acute Hemorrhagic Pancreatitis, Bull. Johns Hopkins Hosp. **58**:212-259 (March) 1936.

stances has been demonstrated by the use of cross circulation in dogs; however, further experimental procedures have shown that the toxicity is dependent on bacterial action. Organisms in healthy pancreatic and hepatic tissues either resemble or are identical with Clostridium welchii.

In considering the etiology of acute interstitial pancreatitis in contrast to that of acute pancreatic necrosis, one observes that the actual trigger mechanism "touching off" the episode is probably somewhat different in the two types, though the blocking of the duct system is the fundamental genetic basis of both. The blocking is temporary and recurrent in the former, tending to increase in severity, whereas in the latter there is a more permanent obstruction, resulting in the more dramatic climax. Perhaps in a large proportion of cases of the transient condition there is a functional spasm of the sphincter of Oddi, and in the necrotic type there is a more permanent mechanical obstruction secondary to cholelithiasis or some similar condition.

Pathology.—Data with regard to the pathologic changes observed in acute interstitial pancreatitis are limited, because it is exceptional for this lesion to be seen at the autopsy table. Most of the gross findings have been recorded from direct observations during a laparotomy, and most of the microscopic descriptions have been made possible by removal of tissue for biopsy during operation. As early as 1898, Körte [20] described several patients with acute symptoms referable to the upper part of the abdomen in whom surgical exploration was done and in whom the only abnormality found was edema of the pancreas. Later, others, both in Europe and in America discussed these conditions, so frequently confused with intestinal obstruction and other abdominal emergencies, in an effort to clarify the issue. Elman [3] reviewed the literature, added his own observations and proposed recognition of an entity which he termed acute interstitial pancreatitis. In this condition the gland appears tense and almost glistening as though engorged with fluids, and on palpation this sensation of tension is confirmed by a peculiar hardness, almost akin to the "woody" character of malignant tumor. Examination will explain the relative ease with which the glandular portion of the common bile duct might be compressed in the process. It should be emphasized that these changes may not be observed if the patient is operated on after acute symptoms have subsided. There may be fat necrosis limited to the peripancreatic tissues without actual pancreatic necrosis. Further exploration of the biliary system will in many cases reveal some disease, usually chronic cholecystitis or cholelithiasis. Microscopically one finds a gland which is fairly intact, in marked contrast to the cellular holocaust observed in pancreatic

20. Körte, W.: Die chirurgischen Krankheiten und die Verletzungen des Pankreas, Stuttgart, Ferdinand Enke, 1898, p. 175.

necrosis. The acini remain structurally distinct, and the inflammatory nature of the disease is seen in the leukocytic invasion of the interacinar and interlobular tissues. The infiltration is confined in great part to the framework of the pancreas, though frequently there are inflammatory cells and débris within the ducts. Polymorphs are the predominating cells involved. This pathologic process is to be differentiated from chronic pancreatitis, in which fibrosis of the interacinar tissues plays the predominant role, though some pathologists consider the latter change to be sequential to the former.

Acute pancreatic necrosis is truly an exact and descriptive term, as any surgeon who has inspected and palpated the pancreas in such a condition will agree. The gland may lose all semblance of its normal anatomic structure and stand out as a necrotic mass surrounded to a greater or lesser degree by adjacent viscera in an attempt at walling off the process. In contrast to the interstitial type, the pancreas is soft, dark red, purplish or even black, depending on the pathologic stage. There are varying degrees of fat necrosis, which is scattered widely over the pancreas or the surrounding omentum and mesentery. On opening the peritoneal cavity the surgeon is confronted with free brownish fluid, formed by increased peritoneal transudation. This fluid is often mixed with particles of necrotic material.

Under the microscope these sections of necrosis are distinctive in their utter lack of structure. The acini are destroyed and about the areas are zones of polymorphic infiltration. According to Gatewood,[21] this infiltration and swelling of the gland are responsible for the severity of the pain produced. Little more than this can be stated about the histopathologic picture of this condition.

Therapy.—There is no necessity for any type of immediate surgical intervention in cases of acute interstitial pancreatitis; the patients, therefore, fall into the group which may be safely watched. The therapy is symptomatic until the acute symptoms subside, at which time one may resort to prophylactic surgical intervention in an effort to prevent recurrence. As the biliary system appears to be the most frequent offender, most surgical procedures consist of cholecystectomy, provided, of course, that there is cholecystographic evidence of a pathologic gallbladder. At operation the common duct may be explored (often via the cystic duct) and drainage of bile through the cystic duct carried out. In conjunction with exploration of the common duct, probes may be passed into the duodenum, actively dilating the sphincter.

Symptomatic therapy during the acute phase of acute interstitial pancreatitis consists in the administration of carbohydrates either by

21. Gatewood: Acute and Chronic Pancreatitis, S. Clin. North America **17**: 473-487 (April) 1937.

mouth or parenterally in sufficient quantities to protect the liver, which is so often involved. Sedation plays an important role in the comfort of the patient and may even prevent secondary attacks brought about by nervousness and apprehension. Amyl nitrite has been suggested recently to relieve spasm of the sphincter of Oddi. Morphine is of little value and may actually increase the intensity of the pain or precipitate another attack.

About the therapy of acute pancreatic necrosis there is much controversy. Finney [14] prescribed early laparotomy and drainage as the method of choice. On the other hand, Smead [22] questioned the wisdom of early operation with wide exposure, incision and tamponade of the pancreas and drainage of the biliary system, favoring a delay of several days or even a week or two.

There are also extreme differences in the reported mortality of acute pancreatic necrosis. Although in most statistics the rate ranges around 50 per cent or higher, Mikkelsen [23] between the years 1926 and 1934 treated 30 patients with acute pancreatitis with a mortality rate of only 7.5 per cent. He expressed opposition to early surgical intervention. De Klimkó,[5] to whose series of cases I have already referred, agreed that after immediate operation there was a mortality rate of 90 per cent, whereas after delayed operation the rate fell to 12 per cent. While statistics such as these may seem convincing, they must be analyzed and only patients with true pancreatic necrosis considered. Unless this is done, mortality statistics are valueless, because they include instances of acute interstitial pancreatitis, which it is now known will subside spontaneously.

It appears from the literature thus briefly cited that the consensus bears out the belief that early surgical intervention in cases of acute pancreatic necrosis is not advisable and that operation should be delayed. That indefinite delay is not the proper course, however, is indicated by the present series, in which such a policy resulted in the death of all 5 patients.

SUMMARY

Ten cases of acute pancreatitis are reported, segregated for further analysis into two groups of 5 cases each. Investigation reveals differences which are significant if the condition called acute pancreatic necrosis is to be recognized; it is in cases of this condition that conservative therapy must be replaced by other treatment, notably laparotomy, with a view to reducing the exceedingly high mortality. In this series,

22. Smead, L.: Treatment of Acute Pancreatic Necrosis, Am. J. Surg. **32**: 487-497 (June) 1936.

23. Mikkelsen, O.: Pancreatitis acuta: Schwere Fälle, besonders hinsichtlich ihrer konservativen Behandlung, Acta chir. Scandinav. **75**:373-415, 1934.

all patients with this condition died after conservative therapy. Operation for drainage of the lesser peritoneal cavity has long been the accepted procedure and is based on the favorable effect of allowing active trypsin an exit, thus minimizing its destructive action on the pancreatic and surrounding tissues. Preparatory measures, such as transfusions and administration of fluids, must be used in view of the shock which is so frequently present. Doubtless the mortality of this disease will always be high, but on the basis of this report a change from conservative therapy is indicated. A reasonable procedure would seem to be treatment by conservative measures during a preparatory period, followed by operation as soon as possible. Patients suffering from the transient type of interstitial pancreatitis recover promptly; the symptoms of those harboring a necrotic pancreas do not automatically subside. Patients can be converted into better risks during the period of observation; this will reduce the high mortality of laparotomy.

CONCLUSION

Five patients with acute interstitial pancreatitis were treated conservatively, and all recovered; 5 patients with acute pancreatic necrosis were similarly treated, and all died. Both types of disease can be diagnosed by early determination of the value for blood amylase. The clinical differential diagnosis of the two conditions is discussed; this is important in order to reduce the mortality of the second condition, acute pancreatic necrosis. It is suggested that in cases of the latter entity operation be carried out as soon as the diagnosis is made provided the patient can be made operable by appropriate preparatory procedures.

EFFECT OF SCLEROSING SUBSTANCES ON HEALING OF FRACTURES

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AND
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It is not within the scope of this paper to discuss the causes of delayed union or nonunion of fractures. It may be briefly mentioned that local as well as constitutional factors come into consideration and that in spite of careful attention to these conditions frequently no union can be obtained. The field for an efficient stimulant of regeneration of bone is open.

While the humoral theory of osseous growth finds numerous proponents, another school of thought is gaining popularity; this school teaches that bone is formed by direct action of specific cells. Although the problem is still awaiting solution, the cellular theory is supported by the results of many experiments and by many clinical observations. It is possible, therefore, that a stimulant of osseous growth which can be used in selected cases of nonunion or delayed union may be found. Such an agent must fulfil the following conditions: (1) It must not cause any injurious local effects or a systemic reaction; (2) it must be sterile, and (3) its injection must be followed by a painless therapeutic response.

Our attention has been attracted to the use of sclerosing solutions in the treatment of hernia. The rationale of this procedure has been firmly established, since it has been shown that the solutions used for injection cause a proliferation of tissues, which gradually closes the hernial ring. The therapeutic effectiveness of injection of sclerosing substances in selected cases of hernia induced Schultz [1] to employ it in treatment of subluxation of the temporomandibular joint. This use of the procedure was successful.

Bone is modified connective tissue; wherever and whenever bone is formed, the process starts with undifferentiated mesoblastic cells and culminates in transformation of the mesodermal tissue into the bone

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1. Schultz, L. W.: Treatment for Subluxation of the Temporomandibular Joint, J. A. M. A. **109**:1032 (Sept. 25) 1937.

under the influence of local or systemic stimuli. The following experiments were undertaken with the hope that injection of sclerosing substances would stimulate regeneration of bone.

EXPERIMENTAL METHOD

Three substances were selected for the experiments: proliferol "B," proliferol "T" and sylnasol.[2]

In 24 rats under ether anesthesia, fractures were produced manually in the center of the right tibia. Two days later, 0.125 cc. of proliferol "B" was injected at the site of fracture in 6 rats; 6 animals received a double dose of the same solution; 6 rats were given injections of 0.125 cc. of physiologic solution of sodium chloride, and 0.25 cc. of the same saline solution was injected into each of the remaining 6 rats. The injections were repeated every second day to a total of twelve. One animal of each group was killed eight, twenty-one, twenty-eight, forty and seventy days after the first injection. Roentgenograms of the fractured extremity were taken immediately after the animal had been killed; the extremity was then carefully dissected and placed in solution of formaldehyde U. S. P. for microscopic studies. After decalcification of the bone, serial sections were made longitudinally through the site of fracture.

In a series of identical experiments proliferol "T" was used, and in the third series sylnasol was used.

RESULTS

The first roentgenograms, taken ten days after the production of the fracture, or eight days after the first injection, showed that all fractures were produced in practically the same portion of the tibia. The second series of pictures, taken twenty-three days after the production of the fracture, or twenty-one days after the first injection, showed beginning callus formation; the position of the fragments was not, of course, identical in all the animals. The third series of pictures, taken thirty days after the production of the fracture, or twenty-eight days after the first injection, showed more advanced callus formation, with resulting increase in the density of the shadows. The last series of pictures was taken seventy-two days after the production of the fracture, or seventy days after the first injection. Firm consolidation of the fragments was

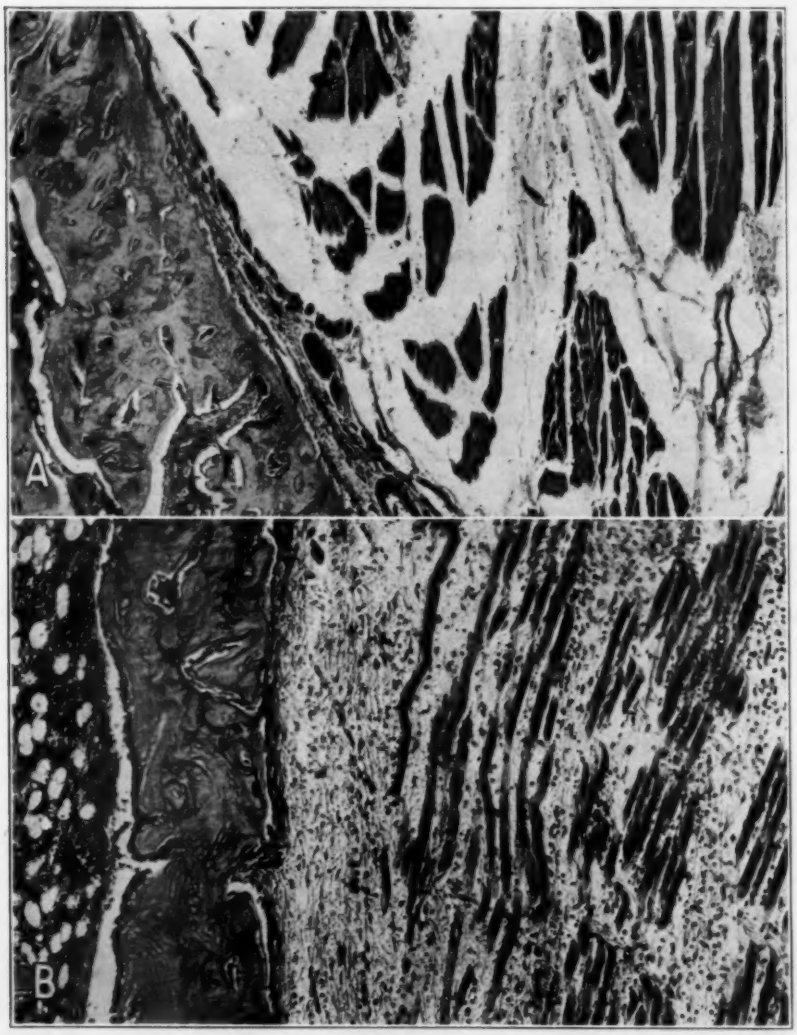
2. According to the manufacturer, proliferol "B" is a distillate of several botanic drugs of known proliferating properties, containing thymol and 0.5 per cent tannic acid. Proliferol "T" is a mixture of 7 parts of proliferol "B" and 1 part of thuja injection fluid. (Thuja injection fluid, or thuja mixture, consists of 50 parts phenol, 25 parts alcohol and 25 parts Lloyd's specific tincture of thuja.)

Sylnasol, formerly known as sylasol, is a 5 per cent solution of the sodium salts of certain of the fatty acids of the oil extracted from a seed of the psyllium group.

These preparations have not been accepted by the Council on Pharmacy and Chemistry of the American Medical Association.

Proliferol "B" and proliferol "T" were supplied by the Ulmer Pharmacal Company, Minneapolis; sylnasol, by G. D. Searle & Co., Chicago.

revealed. A comparison of the pictures belonging to the same series at no time disclosed any differences between the fractures treated with proliferol "B," those treated with proliferol "T," those treated with sylnasol, those treated with saline solution as far as the time of the



A, photomicrograph (×94) of a section of a rat's leg twenty-one days after production of a fracture of the tibia followed by injections of saline solution. The tissue at the left of the specimen is bone; that on the right, muscular tissue. There is no evidence of an inflammatory reaction. *B*, photomicrograph (×94) of a section of a rat's leg twenty-one days after production of a fracture of the tibia followed by injections of proliferol T. The tissue at the left of the specimen is bone; that on the right, muscular tissue. Numerous fibroblasts are scattered between the muscular fibers.

first appearance of the callus, the density of the shadows or the ultimate consolidation of the fragments was concerned.

As can be seen in the photomicrographs, twenty-one days after the first injection there were no signs of an inflammatory exudate after injection of proliferol "T"; in comparison with the fracture treated with injections of saline solution, however, there was a marked increase of fibroblasts around the bone and between the bundles of muscles. Similar findings were made in the fractures treated with proliferol "B" and in those treated with sylnasol. No considerable differences could be detected in the effect of the three sclerosing substances except that the amount of newly formed connective tissue seemed to be larger around fractures treated with proliferol "T" than around those treated with the other two sclerosing substances. Histologic studies of specimens obtained at longer intervals after the first injection showed increase and condensation of the fibrous tissue in the soft parts at the site of fracture.

A study of sections made at various times after the first injection failed to demonstrate any differences in the size of the callus in the animals treated with injections of saline solution and in those treated with sclerosing substances.

COMMENT

It cannot be stated definitely that the sclerosing substances used in the experiments just described do or do not stimulate callus formation, because various sources of error must be considered. One of them lies in the technic of preparation of sections: it is obviously impossible to make comparable sections at the site of fracture in the different animals used for experiment, and the deviations of direction will naturally be responsible for a spurious increase of the size of the callus. Another cause of the apparent failure to stimulate callus formation may be attributed to mechanical factors; after the first two injections the induration of the soft tissues surrounding the fracture made impossible the introduction of the sclerosing solution between the fragments, so that the injected fluid was probably deposited at some distance from the fracture and could not reach its destination.

Although neither the roentgenologic nor the histologic findings could demonstrate stimulation of the callus by injections of the sclerosing solutions, one effect was undoubtedly obtained, that is, formation of new, dense connective tissue around the fracture. This histologic change was responsible for the clinical observation of a rock-hard induration. Such change may offer a certain advantage, serving as "internal splinting" of the fragments. Theoretically a disadvantage could be created by the formation of very dense tissue, with resulting compression of the blood vessels and impairment of blood supply to the site of the

fracture. This, however, was not the case, for the number and size of the capillaries in specimens obtained from fractures treated with sclerosing solutions compared favorably with the number and size of those observed in the control specimens.

SUMMARY

Injections of 0.125 and 0.25 cc. of proliferol "B," proliferol "T" or sylnasol at the site of fracture of the tibia in rats failed to produce roentgenologically or histologically demonstrable stimulation of regeneration of bone.

No untoward local or general effects were observed.

Marked fibrosis followed injections of the sclerosing substances.

CONCLUSION

Although injection of sclerosing substances at the site of fractures of the tibia in rats apparently did not stimulate the rate of regeneration or the amount of the newly formed bone, the resulting fibrosis of the surrounding tissues may have a therapeutic value in selected cases, serving as an internal splint for the bone fragments.

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Dr. Otto F. Kampmeier, head of the Department of Anatomy, and Dr. George E. Wakerlin, head of the Department of Physiology, made helpful suggestions and criticisms concerning this work.

SUNRAY HEMANGIOMA OF THE SKULL

REPORT OF A CASE

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AND
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Hemangioma of the skull is exceedingly rare. A recent thorough review of the literature by Anspach [1] disclosed a total of 21 reported cases. He added a detailed account of a case of his own, emphasizing the special roentgen features. The tumor was first diagnosed as a sarcoma. An operation was attempted but had to be abandoned because of profuse bleeding. However, after fifteen years the patient was still in good health and roentgenograms of the skull showed the characteristic "sunburst" effect of a benign, slowly growing hemangioma.

It is of interest that in 7 of the 21 cases of hemangioma of the skull operation was performed. Brief reference to them follows: In 1877, Ehrmann [2] performed trephination of the skull of a 40 year old woman who had been suffering from severe headaches for many years. She died of meningitis shortly thereafter. At autopsy a soft cavernous hemangioma was seen in the left parietal region, involving the diploë but leaving the inner and outer tables of the skull intact. Zajaczkowski,[3] in 1901, removed a cavernous angioma from the left parietal region of a 38 year old patient who had been aware of the growth for six years. The tumor pulsated synchronously with the heart and was attached to the dura. In 1905, von Bergmann [4] removed a myelogenous hemangioma from the occipital bone by joining three trephine holes which were well outside the limits of the tumor. The tumor measured 3 by 4 by 1 cm. and did not involve the dura.

From the Neurosurgical Service of the Mount Sinai Hospital.

1. Anspach, W. E.: Sunray Hemangioma of Bone, with Special Reference to Roentgen Signs, J. A. M. A. **108**:617 (Feb. 20) 1937.

2. Ehrmann, 1847, cited by Schöne, G.: Ueber einen Fall von myelogenem Hämangiom des Os occipitale, Beitr. z. path. Anat. u. z. allg. Path., 1905, supp. 7, p. 685.

3. Zajaczkowski, A.: Ein Fall von Angioma cavernosum des Stirnbeines, abstracted, Centralbl. f. Chir. **28**:507, 1901.

4. von Bergmann, cited by Schöne, G.: Ueber einen Fall von myelogenem Hämangiom des Os occipitale, Beitr. z. path. Anat. u. z. allg. Path., 1905, supp. 7, p. 685.

Complete removal of a hemangioma of the skull was reported by Cushing.[5] The tumor was regarded as a melanotic sarcoma until three years after the operation, when the specimen was again studied and the final diagnosis of cavernous hemangioma made.

Dikansky[6] reported 2 cases of cavernous hemangioma of the skull in which recovery followed operation. One of the patients had intractable headaches, vomiting and convulsions associated with unconsciousness. Bucy and Capp[7] have clearly demonstrated that "sunburst" trabeculations are characteristic features of the roentgen picture of hemangioma in a flat bone. However, we find no report of a correct diagnosis prior to the successful removal of such a tumor from the skull. In the case to be described a hemangioma of the skull was diagnosed preoperatively.

REPORT OF A CASE

History.—On Aug. 3, 1937, a 47 year old white woman, a housewife, was admitted to the hospital because of a growth in the left parietal region. Her health was otherwise excellent, and her past medical history had no relation to the present complaint. She recalled that on a number of occasions in the past few years she had bumped her head against a closet projecting above the kitchen sink, usually striking the site of the present lesion. She had first become aware of the tumefaction six months before admission. Since that time there had been a gradual increase in the size of the growth. There was no pain or discomfort except for a dull ache when pressure was applied to the tumor. The patient sought hospitalization because she was greatly concerned about the possible malignant character of the growth and the danger that it might lead to cerebral involvement.

Examination.—The patient was well nourished and well developed. She was ambulatory and was not in acute distress. Over the left parietal region was a hard, bony protuberance about 3 cm. in diameter, which seemed to merge into the bones of the skull. Firm pressure in the region of the protuberance caused a vague feeling of discomfort, but there was no local tenderness, pulsation or bruit. The blood pressure was 95 systolic and 65 diastolic. Physical examination otherwise revealed nothing abnormal. Roentgen examination of the skull showed an irregular area of rarefaction in the left parietal region, about 3 cm. in diameter, involving chiefly the outer table of bone. The structure of this area (fig. 1) suggested trabeculations of bone radiating from a common center and had the appearance of a "sunburst" hemangioma.

Course.—The patient was advised that the tumor was benign and self limited. Nevertheless, she remained intensely agitated and feared that possibly the physicians concealed information as to the true nature of the growth. When she became increasingly disturbed emotionally, so that she could not eat or sleep, an operation was thought to be indicated.

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5. Cushing, H.: Surgical End-Results in General, with a Case of Cavernous Hemangioma of the Skull in Particular, Surg., Gynec. & Obst. **36**:303, 1923.

6. Dikansky, M.: Zwei Fälle von Haemangioma cavernosum des Schädels, Deutsche Ztschr. f. Chir. **236**:648, 1932.

7. Bucy, P. C., and Capp, C. S.: Primary Hemangioma of Bone, with Special Reference to Roentgenologic Diagnosis, Am. J. Roentgenol. **23**:1, 1930.

Operation (Dr. Abraham Kaplan).—On August 6, with the patient under the influence of avertin with amylene hydrate and with the use of local anesthesia induced with procaine hydrochloride, a vertical incision was made over the tumor. As the skin and the galea were retracted, the periosteum over the growth was found completely intact (fig. 2). Four burr holes were made about 1 inch (2.5 cm.) from the periphery of the vascular tumor. The burr holes were then joined with

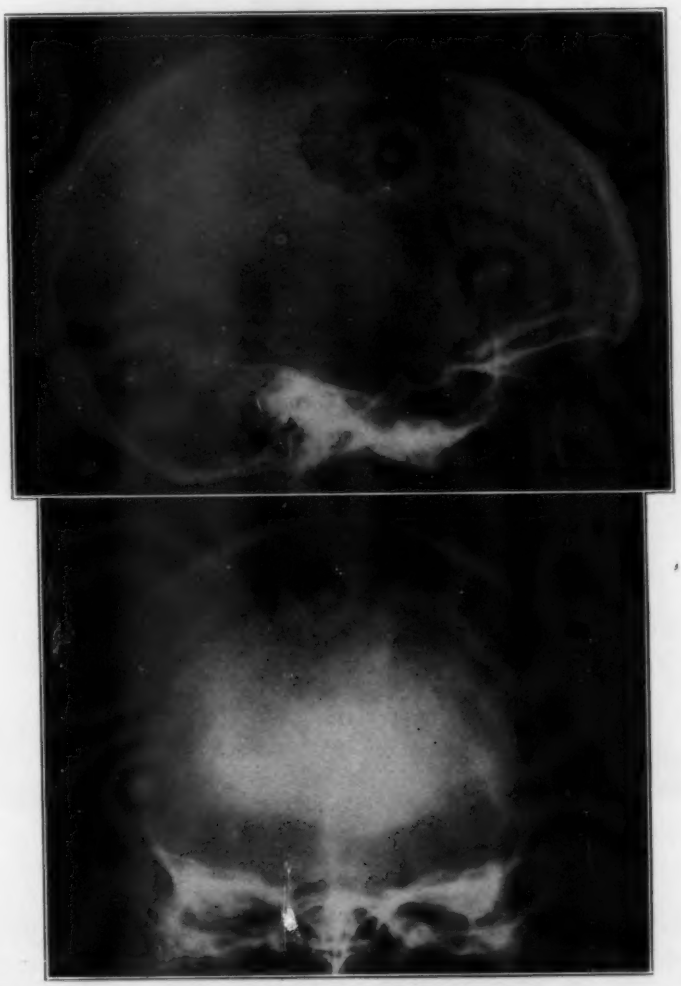

Fig. 1.—Roentgenograms of the skull, showing the characteristic trabeculations of bone radiating from a common center.

a Gigli saw, and the attendant bleeding was controlled with bone wax. As the tumor was elevated, it separated easily from the dura. No sooner was the tumor removed than the troublesome bleeding ceased. The dura was not involved. The pulse and blood pressure remained at a good level, and transfusion was not necessary. Closure of the muscle, galea and skin over the resulting defect afforded natural and secure protection.

Fig. 2.—Outer surface of the tumor, with intact periosteum.

Fig. 3.—Inner surface of the specimen. The periphery shows normal thickness of the skull; the central portion presents an ovoid mottled area of thickening, with fine granular nodulations.

Pathologic Report.—Gross Examination: The specimen consisted of a resected portion of the skull measuring 5 by 4.5 by 2 cm. (fig. 3). The periphery of the resected bone was of normal thickness and appearance. The central portion of the specimen presented an ovoid area of thickening measuring 3.5 by 3.5 by 2 cm., the surfaces of which were mottled blue and yellow, with fine granular nodulations. The tumor mass was well demarcated on the surface, and on section the inner and outer tables were seen to be thinned but intact. Throughout the mass were coarse bony trabeculations which radiated outward from the center.

Microscopic Examination (fig. 4): The bone marrow was completely replaced by fatty angiomatous tissue which surrounded many thin bony trabeculae. The

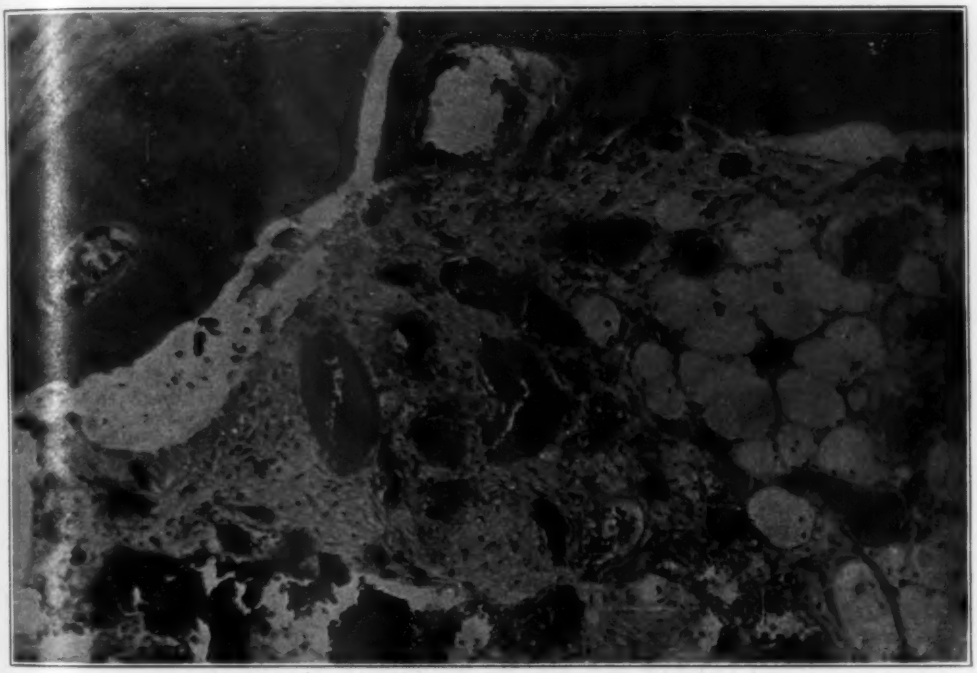

Fig. 4.—Microscopic section of the tumor, showing the intact periosteum and the fatty angiomatous tissue surrounded by many thin bony trabeculae.

periosteum was intact. The stroma consisted of delicate strands of connective tissue accompanied by slender bony trabeculae. There was no evidence of mitosis.

Diagnosis.—A diagnosis of hemangioma was made.

Postoperative Course.—Recovery was uneventful, and the patient was discharged from the hospital on the eighth postoperative day. At the time of writing, one and one-half years after discharge, she is entirely well physically and mentally.

COMMENT

This case illustrates many of the typical features of hemangioma of the skull. Repeated mild local trauma over the site of the tumor is so frequently found in the histories of patients with this lesion that there

appears to be a definite etiologic relation. Although the rate of growth of such a tumor may be slow, it eventually becomes so large and so vascular that operative intervention is hazardous. The subjective complaints, though insignificant at first, steadily increase with the growth of the neoplasm. Headaches become more frequent and may be associated with vomiting or even with convulsions and unconsciousness.

Histologic studies show that a hemangioma grows slowly in the diploë of the skull, arising from a center and radiating toward the inner and outer tables. As growth continues, trabeculations are formed in the bone, which give the characteristic "sunburst" appearance in the roentgenogram. The cortex of the bone may be destroyed, but the periosteum remains intact. The tumor may undergo cystic degeneration and begin to pulsate, at which time even a slight trauma may be followed by serious complications. Most often such a tumor has been thought clinically to be sarcoma. There is some congenital disposition to such growth,[8] and it may be associated with hemangiomas in other organs.[9]

Increasing familiarity with the characteristic roentgen picture of sunray hemangioma makes it probable that the correct diagnosis will be made earlier and with greater frequency. The mental and physical symptoms as well as the cosmetic effects resulting from this usually benign tumor may make operative intervention advisable. Although roentgen therapy may be used as an alternative procedure, Bucy and Capp [7] have found that excision of the hemangioma usually provides the earliest and best results. If excision is done outside the border of the tumor and the surgeon is prepared to control the troublesome bleeding, there should be little difficulty or risk in removing the growth.

SUMMARY

A case of sunray hemangioma of the skull is presented. This rare type of tumor can now be diagnosed preoperatively. The typical clinical roentgen and therapeutic features are discussed, and the surgical aspect is briefly reviewed.

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SULFAPYRIDINE IN TREATMENT OF PNEUMONIA, WITH SPECIAL REFERENCE TO POST-OPERATIVE PNEUMONIA

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No report has come to our attention concerning the effect of sulfapyridine (2-[paraaminobenzenesulfonamido]-pyridine) on postoperative pneumonia. We wish to record our experience with this drug in 21 cases of postoperative pneumonia and 6 cases of primary pneumonia. This includes the cases of all patients with uncomplicated pneumonia under our personal supervision to whom we have given the drug up to the time of writing this report.

DOSE

Patients usually were given 15 grains (1 Gm.) of sulfapyridine by mouth every four hours day and night (90 grains, or 6 Gm., per day). The first dose, and sometimes the second dose also, was doubled, making a total of either 105 or 120 grains (7 or 8 Gm.) during the first twenty-four hours. The duration of treatment is indicated in the charts.

UNFAVORABLE EFFECTS

No seriously bad results could be attributed to treatment with sulfapyridine. Significant leukopenia was not observed. Marked cyanosis was not encountered, and there were no cases of hemolytic anemia, drug rash, drug fever or other serious complications.

Approximately half (15) of the patients treated complained of nausea. About half (8) of these 15 were troubled with vomiting sufficiently severe to persuade us to shorten the contemplated course of treatment. In no instance was it necessary to deny the patient needed treatment because of vomiting. Trouble was lessened when the drug was given with milk or other food. Severe nausea was minimized by inhalation of pure oxygen through a nasal mask for one to two hours after each dose. It must be emphasized to the patient, the nurse and the physician that the occurrence of nausea is not justification for discontinuing the use of this drug when it is really needed. Nausea does not indicate a serious toxic response; it is merely an uncomfortable reaction.

From the Division of Medicine, the Mayo Clinic.

THERAPEUTIC RESULTS

The temperature charts (figs. 1 to 8) demonstrate the results. Each dot represents the maximal temperature on one day. The maximal temperature of nearly half of the patients approached normal within twenty-four hours after the beginning of treatment with sulfapyridine. The condition of most of the remainder was significantly improved in

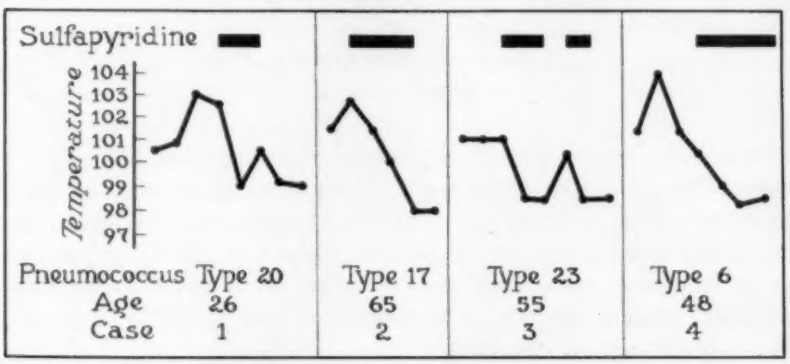

Fig. 1.—Postoperative pneumococcic pneumonia treated with sulfapyridine. In this and in all the following charts each dot represents the maximal temperature for one day. Note the interrupted treatment in case 3 and its relation to fever.

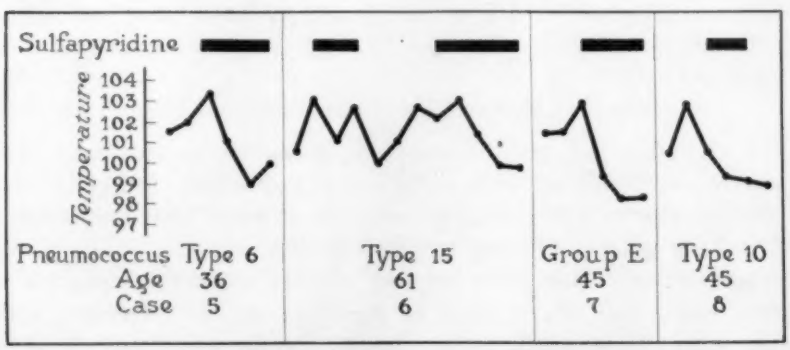

Fig. 2.—Postoperative pneumococcic pneumonia treated with sulfapyridine. Treatment was interrupted in case 6 after secondary closure of the wound; coughing had caused extrusion of viscera. The fever was due in part to localized peritonitis.

forty-eight to seventy-two hours. The results were similar whether or not pneumococci were found in the sputum. Postoperative pneumonia responded as well as primary pneumonia. Older patients responded as well as younger ones. Only 1 death occurred. This was in a case of early fulminating postoperative pneumonia which developed on the

second day after extraperitoneal resection of a carcinoma of the colon (case 12). The patient first received sulfanilamide (total dose, 100 grains, or 6.5 Gm.) and rabbit serum (100,000 units, type 13) on the second day of the pneumonia. Administration of sulfapyridine was started thirty hours before death, and the patient received 135 grains

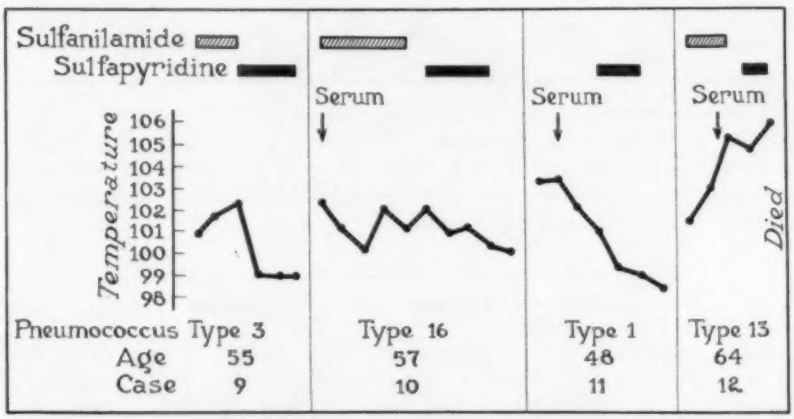

Fig. 3.—Postoperative pneumococcic pneumonia. In each case more than one form of treatment was employed. In cases 10, 11 and 12, 100,000 units each of appropriate antipneumococcus serum was given. Note the apparent effectiveness of sulfapyridine after the apparent failure of sulfanilamide. A roentgenogram taken in case 11 is shown in figure 9 *a*.

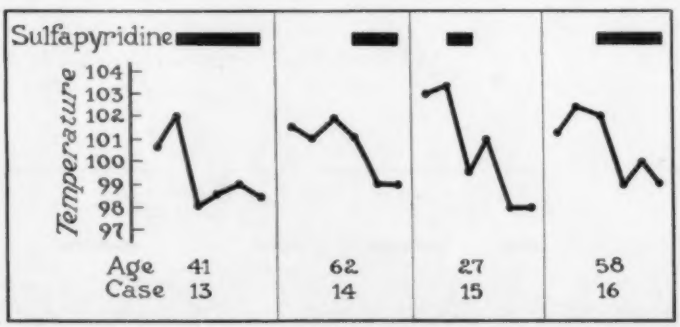

Fig. 4.—Postoperative pneumonia treated with sulfapyridine. Pneumococci were not found in the sputum. A roentgenogram taken in case 13 is shown in figure 9 *b*.

(9 Gm.). As the patient was comatose, the drug was given by duodenal tube. Necropsy disclosed extensive bilateral pneumonia.

In cases 3, 6 and 21 treatment was interrupted. Close correlation between administration of sulfapyridine and reduction of fever was apparent. In case 9 sulfanilamide and sulfapyridine were given at differ-

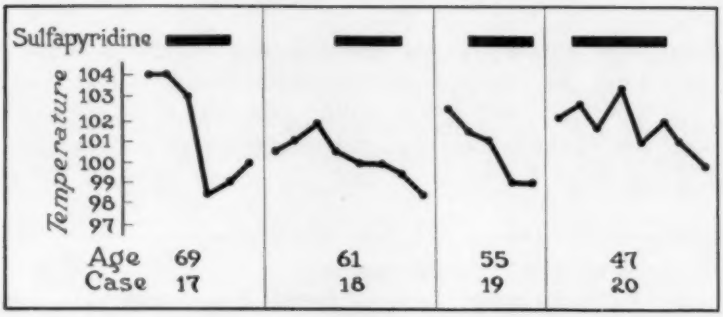

Fig. 5.—Postoperative pneumonia treated with sulfapyridine. Pneumococci were not found in the sputum. Pneumonia was demonstrated roentgenographically in case 20 when treatment was started. Although fever continued for three or four days, evidences of pneumonia, including roentgenographic signs, were absent four days later when treatment was stopped.

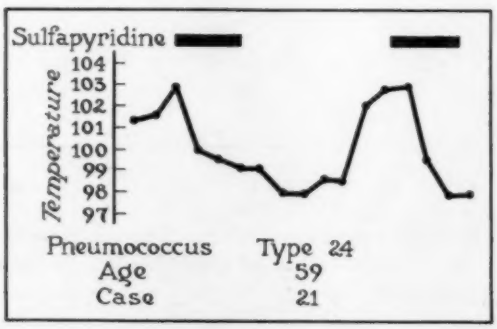

Fig. 6.—Postoperative pneumococcic pneumonia, treated with sulfapyridine. The first course of treatment was for pneumonia of the lower lobe of the right lung (pneumococcus type 24). One week later pneumonia developed in the lower lobe of the left lung, with no pneumococci in the sputum. The response to treatment in both instances was excellent.

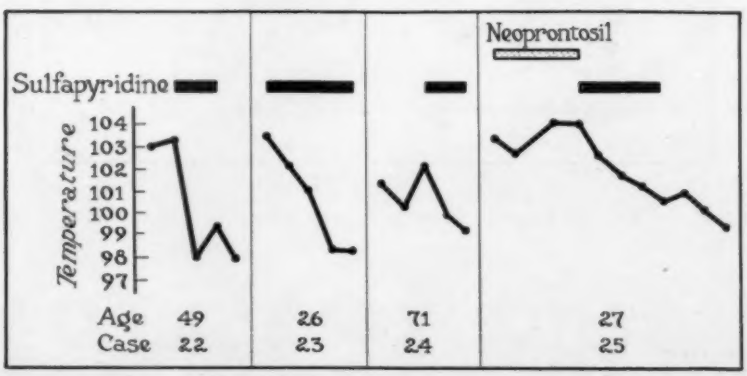

Fig. 7.—Primary pneumonia treated with sulfapyridine. Pneumococci were not found in the sputum. Note that in case 25 sulfapyridine seemed to be beneficial after neoprontosil apparently had failed.

ent times, and an apparent difference between the effectiveness of the drugs was noted. In cases 25 and 26 neoprontosil (administered orally) and sulfapyridine were given, with results similar to those obtained in case 9. In cases 10 and 12 antipneumococcus serum (100,000 units

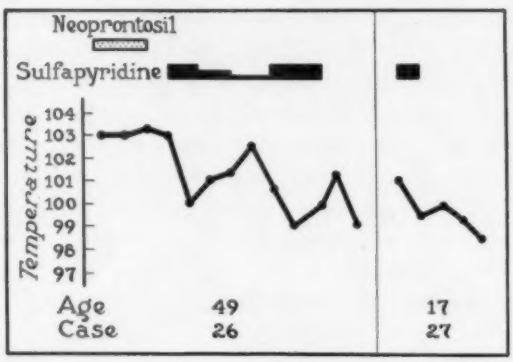

Fig. 8.—Primary pneumonia treated with sulfapyridine. Note that in case 26 reduction of the dose resulted in recurrence of fever but that the fever subsided when administration of the full dose was resumed. Note the apparent failure of neoprontosil.

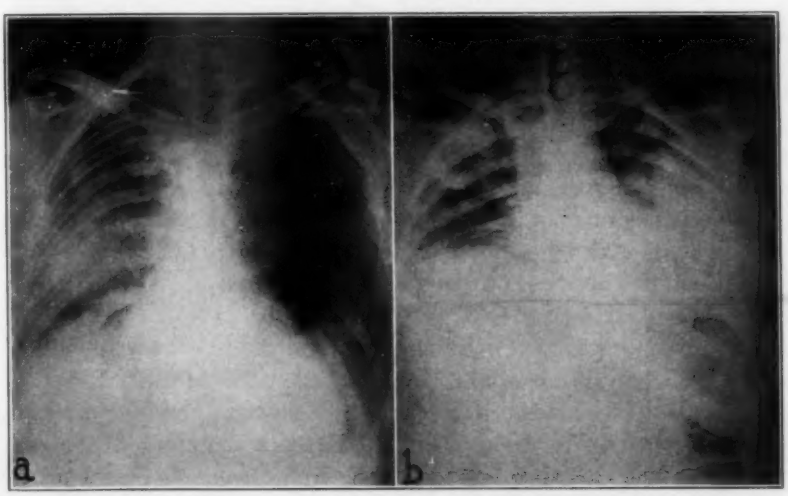

Fig. 9.—Thoracic roentgenogram (*a*) in case 11 and (*b*) in case 13.

in each case), sulfanilamide and sulfapyridine were administered. In case 11 the response to serum therapy (100,000 units) was prompt, but because some degree of fever persisted treatment with sulfapyridine was instituted forty-eight hours after serum therapy had been begun. It must be emphasized that other patients, whose cases are not reported here, responded satisfactorily to serum, sulfanilamide or neoprontosil

and therefore did not receive sulfapyridine. Certain of the charts merely demonstrate that some patients who did not respond to other treatment did subsequently respond to sulfapyridine.

The diagnosis of pneumonia was confirmed by roentgen examination in every case. The roentgenograms taken in 2 of the cases (cases 11 and 13) are shown in figure 9. In cases of mild involvement treatment with sulfapyridine usually was denied if early spontaneous improvement could be anticipated. Sulfapyridine usually was withheld until evidence of serious progressive pneumonia had been obtained. Most patients received nonspecific supportive treatment, including oxygen therapy, hyperventilation with carbon dioxide, intravenous administration of fluids and similar measures. Three patients received positive pressure therapy for pulmonary edema, with successful results. At least half of the patients in the series were so seriously ill that recovery would have been doubtful without sulfapyridine or specific therapy.

COMMENT

Surgeons will welcome this evidence that postoperative pneumonia frequently is arrested by administration of sulfapyridine.

Efforts to avoid atelectasis and aspiration may prevent, or even abort, very early postoperative pneumonia. If these measures are ineffectual, however, within a day or two the problem becomes one of overcoming an acute pulmonary infection. The organisms responsible for such an infection frequently are pneumococci. Of the 21 cases of postoperative pneumonia, pneumococci appeared to be the causative organisms in 13. Several types were identified, including type I. The organisms were recognized by the Neufeld method of typing sputum.

Sulfapyridine cannot be administered parenterally because of its insolubility. The drug is not available, therefore, to patients who are not permitted oral medication after a surgical operation. This constitutes a distinct disadvantage.

Optimal concentrations of sulfapyridine in the blood are not known. Maximal concentrations of the drug varied from 2.9 to 7.7 mg. per hundred cubic centimeters in our cases. Between these limits no significant difference in therapeutic or toxic effects was noted.

Successful administration of the drug may depend on adherence to several rules: 1. Nausea is not an indication for discontinuing administration of the drug. 2. Frequent administration, preferably every four hours day and night, seems desirable. 3. Favorable response should be shown in twenty-four to forty-eight hours by a sharp decline in fever. 4. Prolonged administration is not usually necessary and may be dangerous.

The prognosis of pneumonia is often dependent on the age of the patient. Most therapeutic measures diminish in efficacy as age increases.

Available information appears to indicate that administration of sulfapyridine may be an exception to this rule. More than half of our patients with postoperative pneumonia were 55 years of age or more. Only 4 patients were less than 45 years of age. The age of each patient is recorded on the charts.

SUMMARY

Sulfapyridine may promptly arrest the progress of postoperative as well as primary pneumonia. It may be successful when other chemotherapeutic agents apparently have failed. It is effective in the treatment of elderly as well as the young patients. Pneumococci are frequently the predominant organisms in the sputum of patients with postoperative pneumonia. The drug appears to be equally effective when pneumococci are not identified in the sputum.

Dr. D. F. Robertson, Associate Medical Director, Merck & Co., Rahway, N. J., supplied the sulfapyridine used in this study.

CHANGING EXPERIENCES WITH BENIGN AND MALIGNANT LESIONS OF THE COLON AND OF THE RECTUM

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During the first sixteen years of my experience as an associate of Dr. Bloodgood, I took part in the diagnostic study and operation in 276 cases of lesions of the colon and of the rectum. In addition, I studied sections and roentgenograms in 125 cases of of similar lesions treated elsewhere, in which material was sent to the clinic for diagnosis. Table 1 summarizes these experiences.

In 136 of the 276 personally observed cases, approximately 50 per cent, and in 72 of the 125 cases in which we were sent material, approximately 60 per cent, cancer was present. In the former group, in the great majority of instances the cancer had reached the stage in which one could make the diagnosis by digital palpation and by inspection through a proctoscope when the rectum was involved or by study of a roentgenogram taken after a barium sulfate enema when the lesion was situated higher. Consequently, biopsy was seldom performed. For cancer of the colon (exclusive of the cecum) and for cancer of the rectum, appendicostomy or cecostomy has usually been the first treatment. In the cases of operable cancer the involved segment of bowel was later excised in one or two stages, usually with restoration of the continuity of the colon when the lesion was proximal to the lower portion of the sigmoid and usually with permanent colostomy when the cancer involved the rectum or the rectosigmoid junction.

Roentgen therapy alone or before or after operation was used in 12 of the 49 cases of cancer of the colon and in 27 of the 86 cases of cancer of the rectum, and there is no evidence that it prolonged life, although there is no question that it added materially to the comfort of the patient in a number of instances. In 2 cases of cancer involving the lower part of the rectum the treatment was with radium and roentgen rays. Later I shall refer to these 2 cases in connection with 2 recent cases of cancer of the lower part of the rectum so treated.

The lesion was in the colon in approximately 36 per cent and in the rectum in approximately 64 per cent of the group of 140 patients with benign lesions of the colon and rectum examined in the clinic. Of

the 53 cases in which material was sent to the clinic for diagnosis the colon was involved in approximately 43 per cent and the rectum in approximately 57 per cent (table 1).

Congenital malformations, ptosis, postoperative adhesions, amebic dysentery, colitis and tuberculosis accounted for the great majority of benign lesions of the colon, and in 42 of the 50 instances in which the patients were under our own observation there was no operation. In 4

TABLE 1.—*Lesions of the Colon and of the Rectum (1919 to 1935)*

Lesions clinically observed	276 (colon, 100; rectum, 176)
Cancer	136 (colon, 50; rectum, 86)
Benign lesions	140 (colon,* 50; rectum,† 90)
Lesions observed from tissue, sections and roentgenograms	125 (colon, 59; rectum, 66)
Cancer	72 (colon, 36; rectum, 36)
Benign lesions	53 (colon,‡ 23; rectum,§ 30)

* In 8 cases these lesions were treated by operation. Acute obstruction from volvulus was present in 3, mesenteric thrombosis in 1, perforation in 1 and a fecal fistula in the cecum in 1. In 42 cases no operation was done. The conditions were congenital malformations, ptosis, postoperative adhesions, amebic dysentery and colitis.

† In 9 cases a polyp was present. In the remaining 81 cases there were miscellaneous conditions (fissure and fistula in ano, ischiorectal abscess, pilonidal sinus, prolapse of the rectum or proctitis).

‡ In 2 cases the condition was obstruction due to volvulus; in 2 a polyp was present, and in 19 there were various chronic inflammatory lesions, including colitis and pericolitis, ptosis and tuberculosis.

§ In 12 cases a polyp was present; in the other 18 there were miscellaneous conditions.

TABLE 2.—*Lesions of the Colon and of the Rectum (Oct. 21, 1935, to May 21, 1938)*

Lesions clinically observed	56 (colon, 19; rectum, 37)
Cancer	16 (colon, 10; rectum, 6)
Benign lesions	40 (colon,* 9; rectum,† 31)
Lesions studied from tissue, sections and roentgenograms	7 (colon, 3; rectum, 4)
Cancer	4 (colon, 1; rectum, 3)
Benign lesions	3 (colon, 2; rectum, 1)

* In 5 cases these lesions were treated by operation. Acute obstruction due to volvulus was present in 2, polyp of the sigmoid in 2 and diverticulitis of the transverse colon in 1. In 4 cases no operation was done. Simple colitis was observed in 1 of these, ulcerative colitis in 2 and ptosis in 2.

† In 1 of these cases the condition was lymphogranuloma venereum; in the remaining 30, miscellaneous conditions were present (hemorrhoids, fistula, ischiorectal abscess, pilonidal sinus or fissure).

‡ Tuberculosis was present in 1 case and a chronic inflammatory lesion in 1.

of the 8 surgically treated patients with benign lesions of the colon there was acute obstruction from volvulus or mesenteric thrombosis. In only 2 instances, a case of benign polyp of the sigmoid and a case of diverticulitis of the sigmoid, was cancer considered in the preoperative diagnosis.

A large miscellaneous group of the commoner lesions of the rectum composed the majority of benign lesions of the rectum, and benign polypoid tumors visible through the proctoscope accounted for the large minority.

Table 2 is a summary of the experience of my colleague Dr. George A. Stewart and myself with lesions of the colon and rectum in the past two

and one-half years. It is this changing experience that has stimulated me to review our material and to report in abstract 8 recent cases. Cases 1, 2 and 3 are cases of benign lesions of the colon; in cases 1 and 2 there were precancerous lesions of the sigmoid flexure, and in case 3 there were diverticulitis of the transverse colon and multiple diverticulosis of the sigmoid. Cases 4, 5, 6, 7 and 8 are cases of lesions of the rectum. In case 4 the condition was venereal lymphogranuloma, and in cases 5, 6, 7 and 8 it was early operable carcinoma of the lower third of the rectum. This condition was treated by roentgen and radium therapy.

REPORT OF CASES

CASE 1.—H. M. B., a white man aged 46, consulted me Aug. 13, 1936 because he had noticed bright red blood in the stools. He stated that his father had died of cancer of the sigmoid which on exploration had been found inoperable. The patient had first noticed the presence of bright red blood in the stools on two or three occasions about one year before he sought our advice, and again, temporarily, seven months later. So far as he knew, there had been no recurrence until three weeks before he came to the clinic. The quantity of blood at this time was much greater than on previous occasions, and with the blood there was considerable mucus. On four or five occasions during the past three weeks there had been a normal stool, followed in a few minutes by a desire to evacuate the bowel again. The feces of the second evacuation contained a large quantity of mucus streaked with blood. During the previous three weeks there had been a number of attacks of diarrhea, four or five watery stools being passed in twenty-four hours, followed the next day by a normal evacuation of the bowel.

During the preceding two weeks there had been two independent studies by other physicians. These studies included in each instance a proctoscopic examination. In 1 instance a complete gastrointestinal fluoroscopic and roentgen study had been done; in the other, a roentgenogram of the colon had been taken after a barium sulfate enema. The results were reported to be entirely negative. I repeated the proctoscopic examination on two occasions, with negative results. At St. Agnes' Hospital Drs. E. B. Freeman and E. L. Flippin and I noted in the fluoroscopic examination of the colon during a barium sulfate enema a slight irregularity in the distal portion of the sigmoid. A roentgenogram taken immediately afterward showed no filling defect, but the sigmoid loop was distinctly dislocated from its usual position, suggesting to us the presence of adhesions (fig. 1).

On August 15, 16 and 17 I inspected the stools and on each occasion found fresh blood in considerable quantity.

On August 19 I performed appendicostomy and on August 28 resection of the sigmoid portion of the colon, followed by a lateral anastomosis by the Bloodgood method (fig. 2).

The operative findings consisted of adhesions between the lower part of the middle third of the sigmoid and the omentum, and just at this point there was a distinctly palpable, freely movable polypoid tumor about 2 cm. in diameter, which was not visible through the wall of the bowel. I was unable to palpate any other polypi in the colon between the rectosigmoid junction and the splenic flexure. Photographs of the gross specimen (fig. 3) and photomicrographs (fig. 4) show the structure of the tumor.

Fig. 1.—Roentgenogram of the colon after a barium sulfate enema in case 1. Note the dislocation of the sigmoid loop as described in the text. A benign adenomatous polyp was found at the junction of the lower and the middle third of the sigmoid loop.

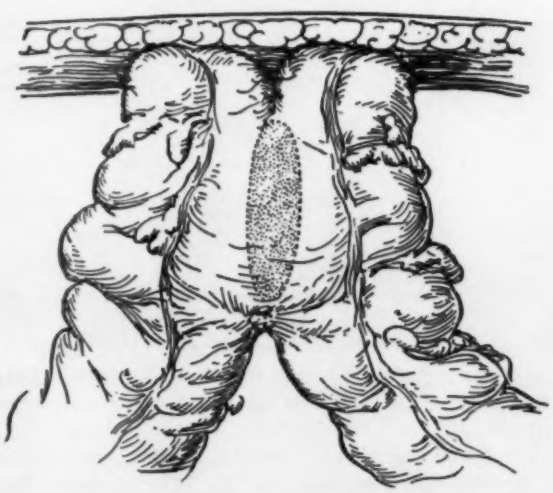

Fig. 2.—Drawing illustrating Bloodgood's method of lateral anastomosis of the colon (from Alexius McGlannan, in Lewis, D.: Practice of Surgery, Hagerstown, Md., W. F. Prior Company, Inc., 1930, vol. 7, chap. 4, p. 105).

On the eighth postoperative day there was a leak to the outside at the site of the anastomosis, and the fistula was still present on the patient's discharge from the hospital, on October 5. In fact, it was not healed until Jan. 18, 1937, three and one-half months later. The appendicostomy wound healed of its own accord, although there was occasional intermittent discharge of cecal contents for some months.

CASE 2.—W. B., a white man aged 47, the brother of the patient in case 1, consulted me on Jan. 26, 1937 because of bright red blood and mucus in the stools for three months. There was slight tenderness in the region of the sigmoid on

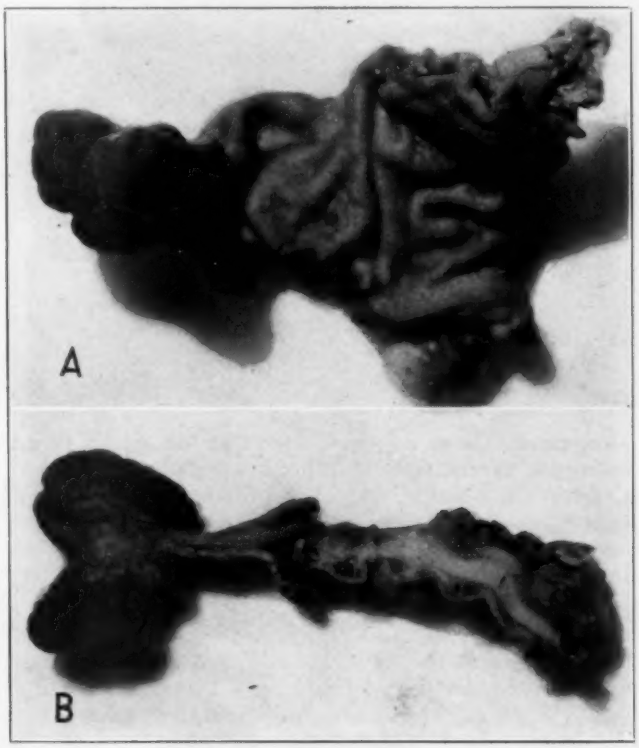

Fig. 3.—Photographs of the gross specimen in case 1, showing the polypoid tumor and the resected segment of the sigmoid.

abdominal palpation. No lesion was present throughout the 24 cm. of the bowel visible through the proctoscope, but mucus and blood were seen running down from the bowel above. A roentgenogram of the colon taken after a barium enema was entirely normal (fig. 5). Six months more elapsed before the patient consented to an operation, notwithstanding the constant presence of blood and mucus in the stools.

On July 9 I resected the sigmoid portion of the colon and made a lateral anastomosis by the Bloodgood method (fig. 2). The operative findings consisted of a freely movable and nonadherent sigmoid containing a palpable polyp approximately 2 cm. in diameter. The polyp was located near the summit of the sigmoid loop. No other polypi were palpable between the rectosigmoid junction and the

splenic flexure. Photographs of the gross specimen (fig. 6) and photomicrographs (fig. 7) show the structure of the tumor.

The operative wound healed per primam intentionem, and the patient left the hospital on July 26, seventeen days after the operation. He returned on August

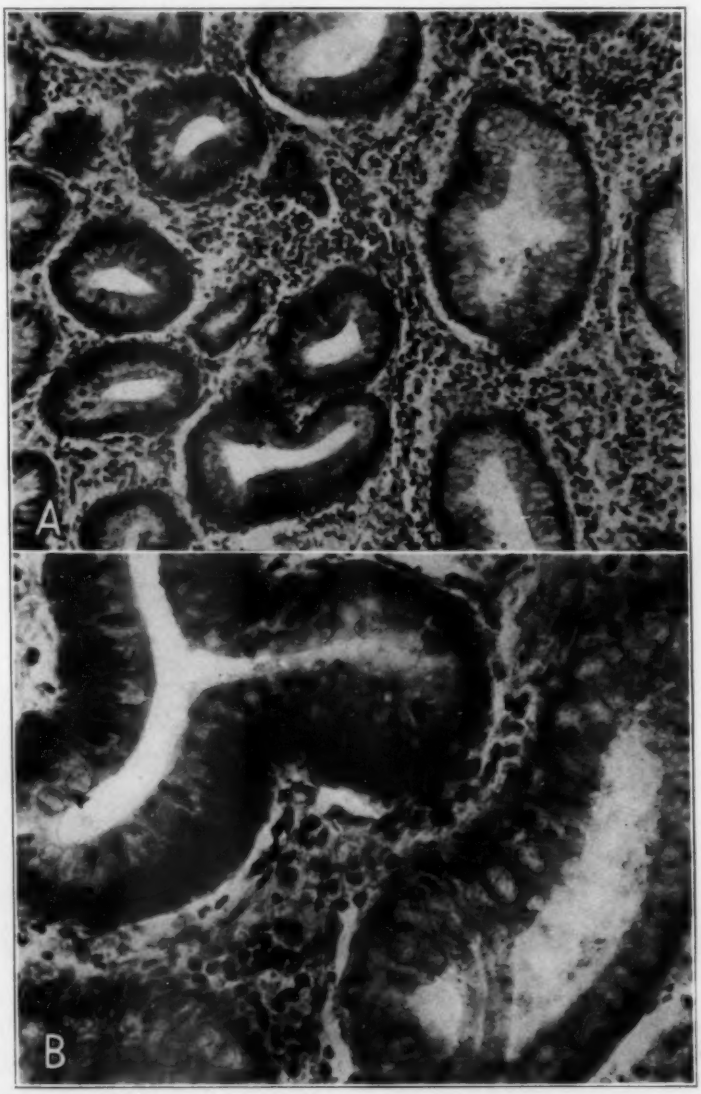

Fig. 4.—*A*, medium low power photomicrograph of the tumor in case 1, showing the adenomatous character of the growth. *B*, high power photomicrograph of the tumor in case 1. Compare the clear cells with small nuclei on the right with the cells containing the hyperchromatic nuclei on the left.

15, twenty days later, because of a small draining sinus in the middle of the scar. This was entirely healed on September 24.

Comment.—The similarity in age, symptoms, duration of bleeding, location, size and gross and microscopic appearance of the lesions in these two brothers was striking. The difference in the appearance of the roentgenograms is accounted for by the presence of adhesions in the first case. The hereditary factor in polyposis intestini has been well established by the studies of Dukes. I am not familiar with any comparable

Fig. 5.—Roentgenogram of the colon after a barium sulfate enema in case 2. The sigmoid loop occupies its normal position. A benign adenomatous polyp was found near the summit of the loop.

studies on solitary polypus of the colon, but it seems that the relation between the sigmoid carcinoma in the father and the presence of a polypoid adenoma of the sigmoid in 2 sons is more than casual. The 2 cases demonstrate the advisability of laparotomy when blood and mucus are constantly present in the stools even when careful proctoscopic and roentgen examinations fail to reveal the site of the lesion. At operation,

search should be made for multiple tumors in the colon. Histologically these tumors are true adenomas and show changes in morphologic and staining characteristics which are definitely precancerous; therefore, even

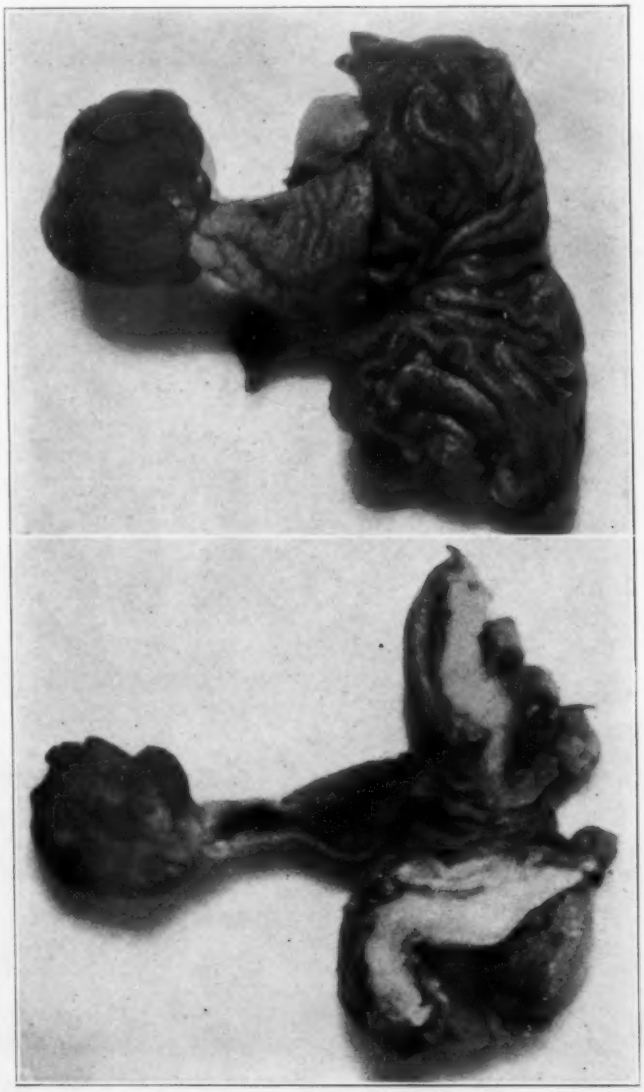

Fig. 6.—Photographs of the gross specimen in case 2, showing the polypoid tumor and the resected segment of the sigmoid.

though they are pedunculated I prefer resection and anastomosis to simple excision. When there are adhesions to the wall of the bowel at the site of the tumor, as in case 1, resection seems almost imperative. Perhaps it is unnecessary to make a preliminary appendicostomy, as

was done in the first case. The advantage of the Bloodgood method is that both ends of the colon can be sutured extraperitoneally between the fasciae, so that if a leak occurs the infection will find its way to the surface more readily.

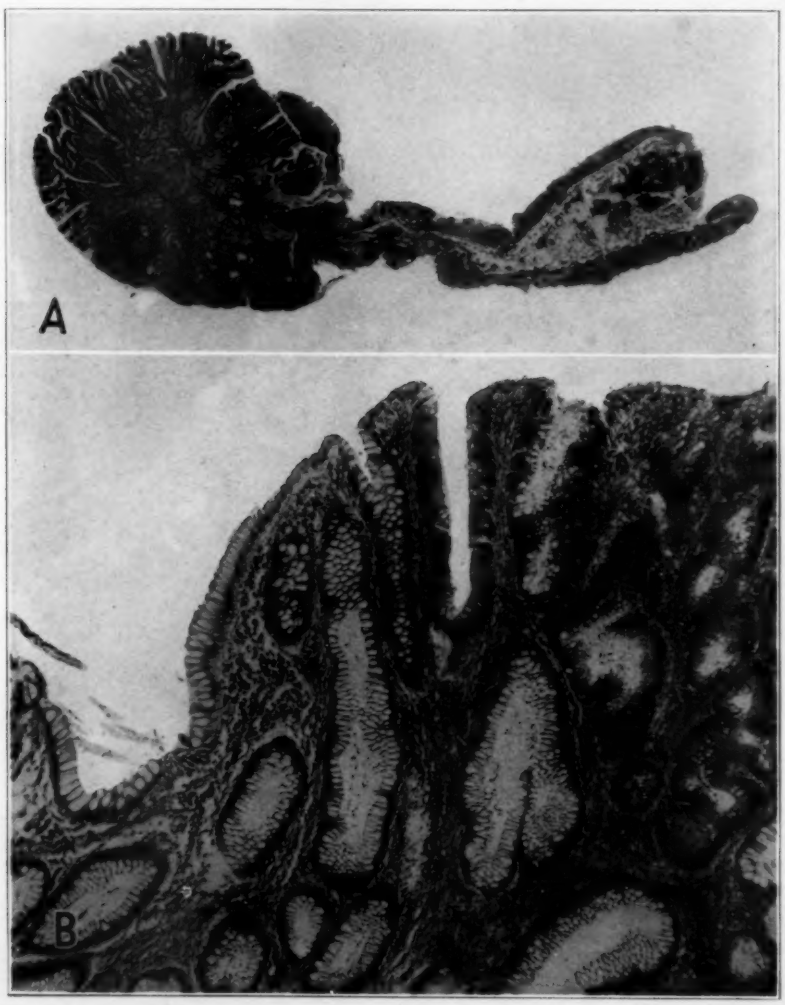

Fig. 7.—*A*, low power photomicrograph of the tumor in case 2, showing the polyp and the pedicle. *B*, medium low power photomicrograph of the same tumor, showing the polyp and the pedicle. Note the adenomatous character of the growth. Contrast the clear cells with small nuclei at the bases on the left with the hyperchromatic nuclei on the right.

Case 3.—R. M., a white man aged 59, consulted us on Dec. 20, 1932 because of pain in the midzone of the abdomen of one month's duration, associated with increasing constipation. Fourteen months previously a calculus had been removed

from the right kidney, and twenty-five years previously appendectomy had been performed.

A roentgenogram of the colon taken after a barium sulfate enema showed multiple diverticula in the sigmoid and a single, larger diverticulum in the right portion of the transverse colon. The symptoms disappeared quickly under treatment with liquid petrolatum and a mild laxative. Roentgenograms of the colon taken at yearly intervals showed no changes in the diverticula during the years 1933, 1934 and 1935 (fig. 8*A*). When the patient returned for examination on Feb. 11, 1937, after a year in Europe, he complained of severe colic across the upper part of the abdomen, of ten days' duration. Palpation elicited tenderness 3 inches (7.5 cm.) to the right of the umbilicus and also below the old appendectomy scar. A roentgenogram of the colon showed a distinct filling defect at the site of the old diverticulum in the right side of the transverse colon (fig. 8*B*). There were no changes in the diverticula in the sigmoid.

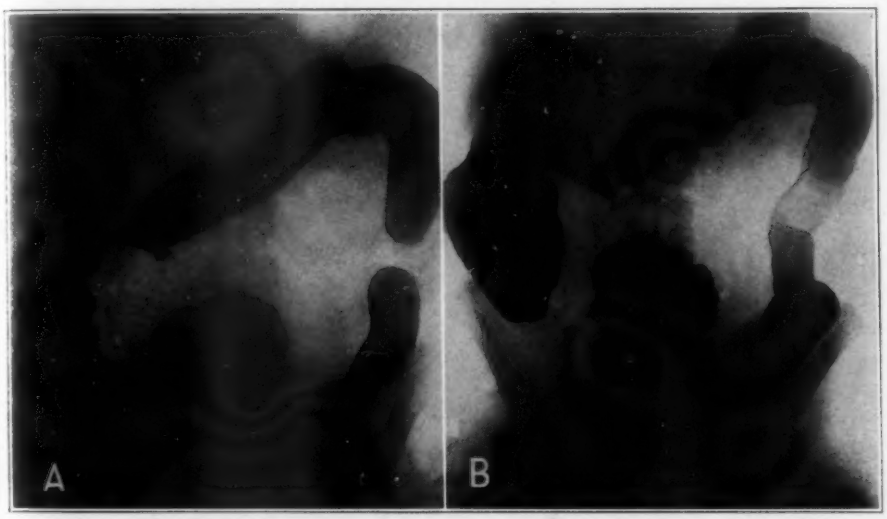

Fig. 8.—*A*, roentgenogram of the colon after a barium sulfate enema in case 3. This picture was taken Dec. 6, 1935. There is a single diverticulum in the right side of the transverse colon and there are multiple smaller diverticula in the sigmoid. *B*, roentgenogram of the colon after a barium sulfate enema in case 3. This picture was taken Feb. 11, 1937, fourteen months after the picture shown in figure 8. There is a distinct filling defect in the right side of the transverse colon at the site of the diverticulum shown in figure 8.

At operation, on February 20, I found a large fatty omentum densely adherent in the right lower quadrant to the cecum and to the parietal peritoneum, probably the result of the old appendicitis. The right two thirds of the transverse colon was dislocated downward, and at the site of the diverticulum the colon was bound by adhesions to the mesentery of the duodenum. A narrow band of adhesions surrounded the colon at this point and constricted the lumen. The diverticulum was encased in a mass of indurated fat, containing calcified nodules.

Exposure of the right half of the transverse colon was obtained by division of the omentum and liberation of the adherent loop by excision of a portion of the

peritoneum of the mesentery of the duodenum together with the indurated fat surrounding the diverticulum. This allowed the colon to resume its normal position, and the division of the band of adhesions encircling the colon at the site of the stricture permitted it to resume its normal shape. The diverticulum was then excised and the stump ligated and inverted (fig. 9 *A*).

On the third day after operation acute parotitis developed on the left side, which rapidly cleared up under roentgen therapy. The abdominal wound healed per priman intentionem, and the patient left the hospital on the thirtieth day after the operation.

When he returned from a trip abroad, on November 15, he complained of occasional abdominal cramps, and examination revealed a small postoperative hernia. The symptoms disappeared in a few weeks under medical treatment, and

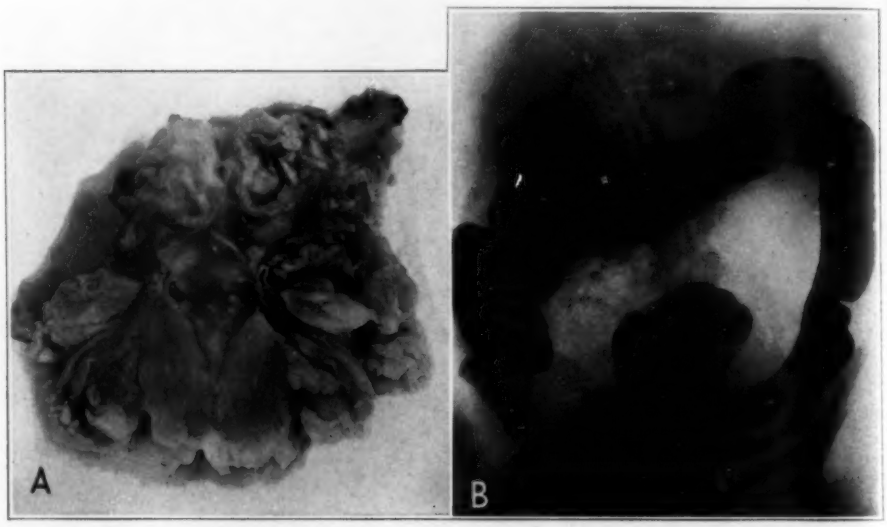

Fig. 9.—*A*, photograph of the bisected gross specimen in case 3, showing (above) the mucosa of the diverticulum and (below) the enveloping mass of indurated fat. *B*, roentgenogram of the colon after a barium sulfate enema in case 3. This picture was taken Nov. 15, 1937, nine months after the operation.

the hernia was controlled by an elastic belt. Figure 9 *B* is a roentgenogram of the colon taken November 15, nine months after the operation.

Comment.—In this case the roentgenogram showing the deformed colon and the filling defect at the site of the preexisting diverticulum indicated diverticulitis rather than carcinoma. However, in a recent case I observed carcinoma at the rectosigmoid junction coexisting with multiple diverticula in the sigmoid, and others have made similar observations. Deforming adhesions may occur in conjunction with diverticulitis, benign and malignant tumors and other conditions, and in the

absence of earlier roentgenograms the preoperative diagnosis may be difficult to establish.

CASE 4.—W. R., a white man aged 47, was admitted to the surgical service of St. Agnes' Hospital through the outpatient department on June 25, 1938. For three months he had noticed diarrhea, tenesmus and the presence of blood and pus in the stools. Examination revealed a distended abdomen. The anus admitted only the tip of the gloved finger. No enlarged lymphatic nodes were palpable

Fig. 10.—Roentgenogram of the colon after a barium sulfate enema in case 4. There is a filling defect from the anus to the rectosigmoid junction, and the colon is distended.

in the groin or elsewhere. A roentgenogram taken after a barium sulfate enema showed a filling defect from the anus to the rectosigmoid junction and marked distention of the colon above (fig. 10). The Wassermann reaction was negative.

On January 27 I made an appendicostomy, and on February 15 I completely excised the rectum by the abdominoperineal method in one stage and made a permanent colostomy. The gross specimen is shown in figure 11, and the microscopic appearance is reproduced in figures 12 and 13. On March 19, Dr. Moses Paulson reported positive cutaneous reactions to two human strains of Frei antigen

at the end of eight days. The patient was discharged from the hospital on April 19. The perineal wound was clean but not entirely healed.

Comment.—My personal experience with venereal lymphogranuloma has been limited, and this is the first time that I have observed this disease in the rectum in the absence of involvement of the inguinal nodes. The absence of metastasis to the regional lymphatic nodes and to the liver notwithstanding the extensive filling defect in the roentgenogram and the large mass palpable from within the peritoneal cavity

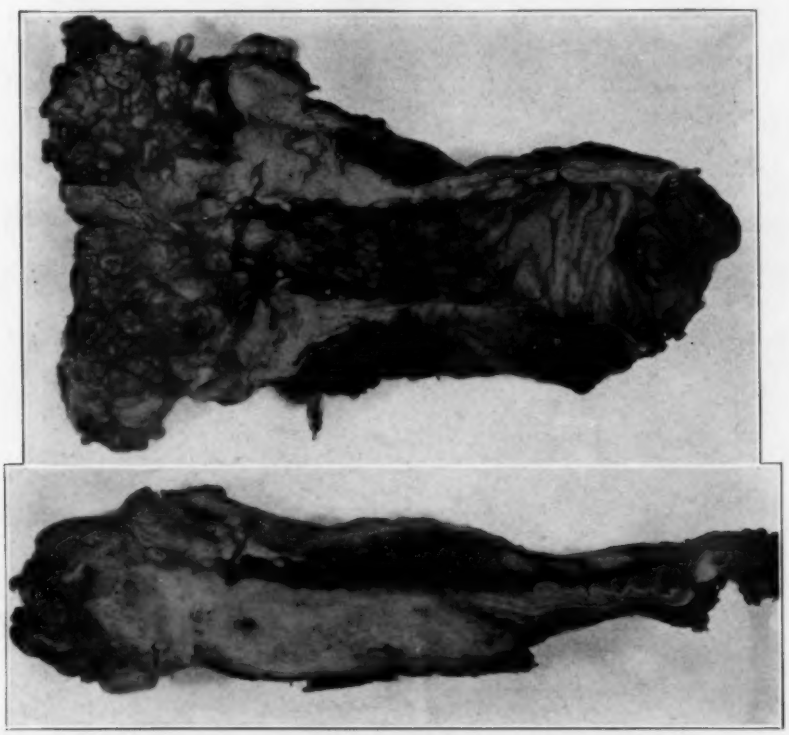

Fig. 11.—Photographs of the gross specimen in case 4. Note the narrow lumen, the ulceration of the mucosa and the thickened wall of the rectum. The fresh specimen had the consistency of leather.

could have suggested to me the possibility of a benign stricture of the rectum. In that event the diagnosis of venereal lymphogranuloma could have been confirmed before instead of after excision of the rectum and the operation confined to the establishment of a permanent colostomy.

Comment.—Cases 5 and 6 were observed with Dr. Bloodgood prior to Oct. 21, 1935 and are the cases to which reference has been made.

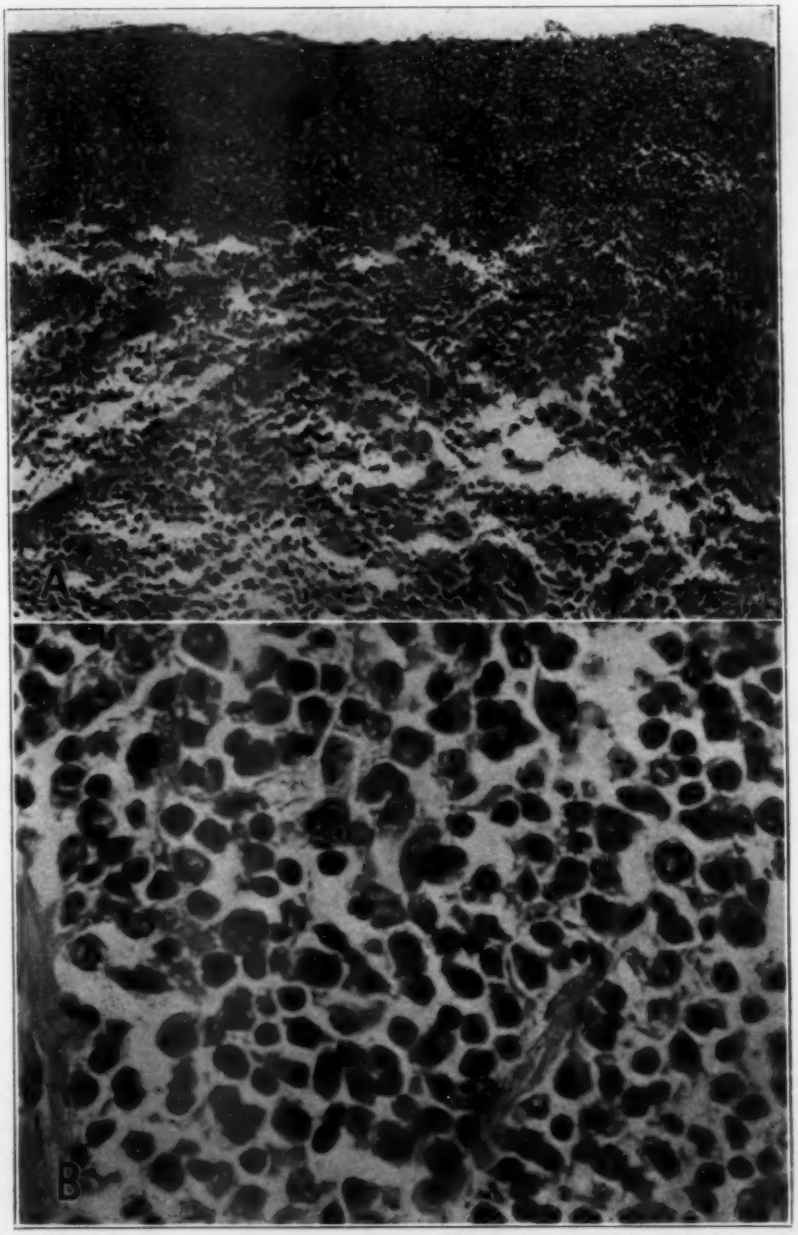

Fig. 12 (case 4).—*A*, low power photomicrograph of the ulcer in the rectum. Note the complete absence of glands, the rich cellular infiltration at the base of the ulcer and the edematous stroma beneath showing plasma cell infiltration. *B*, high power photomicrograph of the rectal wall beneath the base of the ulcer, showing a rich infiltration of plasma cells in the edematous stroma.

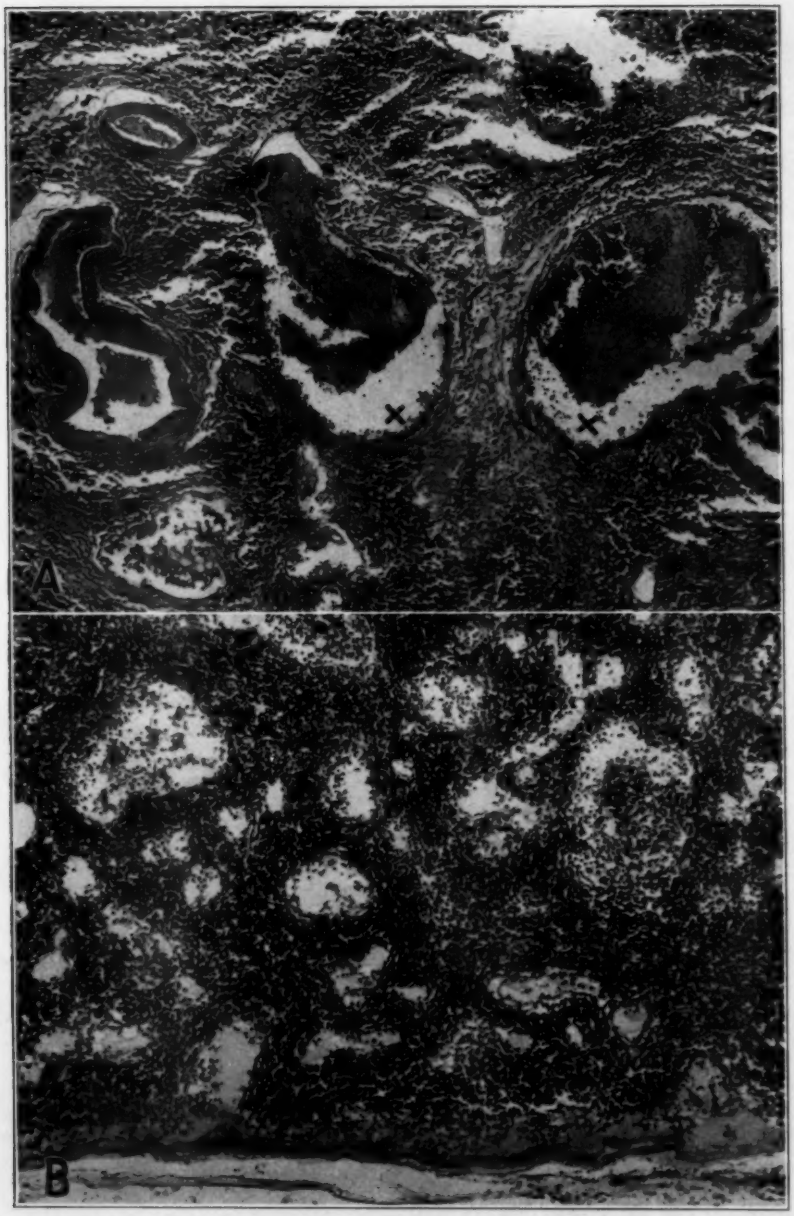

Fig. 13 (case 4).—*A*, low power photomicrograph of the rectal wall beneath the area shown in fig. 12 *B*. Sheets of plasma cells are present as in figure 12. Note particularly the marked dilatation of the lymphatic vessels (x-x). *B*, low power photomicrograph of a lymphatic node. Note the diffuse dilatation of lymphatic vessels.

Fig. 14.—Low power photomicrograph of a frozen section of the tumor in case 5. Note the irregular glands lined by hyperchromatic epithelial cells.

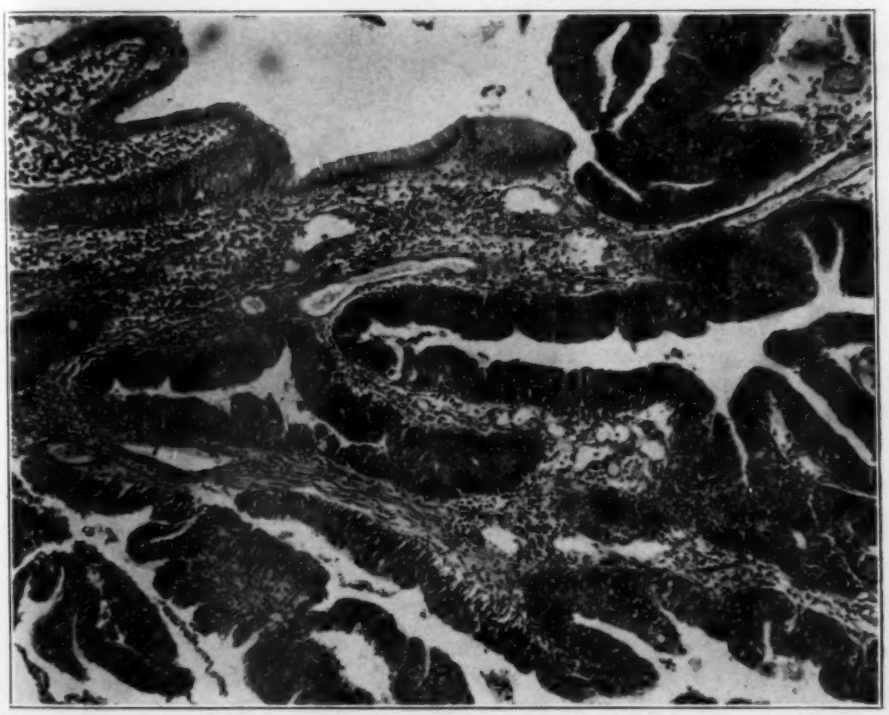

Fig. 15.—Low power photomicrograph of the tumor in case 6, diagnosed as adenocarcinoma. Above and to the left are remains of normal epithelium.

CASE 5.—A. W., a white woman aged 49, consulted the clinic Oct. 23, 1933, bringing with her a section prepared from a bit of tissue removed from the rectum one week previously. The microscopic structure of this tumor is shown in figure 14. She had noticed blood in the stools for two and one-half years. Digital and proctoscopic examinations revealed an indurated ulcer 3 cm. in diameter involving the posterior wall of the rectum, beginning at a point 3 cm. above the sphincter. Dr. Curtis F. Burnam treated the tumor by the daily direct application of radium through a proctoscope. At the examination Feb. 17, 1934 the ulcer was completely healed, and the patient was well on June 6, 1938.

Fig. 16.—Medium low power photomicrograph of the tumor in case 7, diagnosed as adenocarcinoma. Note the adenomatous structure of the tumor, the larger glands (above) lined by goblet cells with the nuclei at the bases and the smaller irregular glandlike structures (below) lined by epithelial cells with hyperchromatic nuclei.

CASE 6.—J. B. F., a white man aged 53, consulted the clinic Feb. 11, 1935 because he had had diarrhea and blood in the stools for three months. Examination revealed an ulcer 6 by 4 cm. in the left lateral wall of the rectum, beginning at a point 3.5 to 4 cm. above the sphincter. The microscopic structure of the tumor is shown in figure 15. Dr. Curtis F. Burnam treated the tumor by the direct application of radium and by external roentgen irradiation. The patient is well.

Comment.—In view of these favorable results, irradiation therapy was tried in the following 2 cases.

CASE 7.—F. L. O., a white man aged 52, consulted the clinic April 3, 1937 because of constipation and blood in the stools. Both symptoms had been present for fourteen months. Hemorrhoidectomy had been performed six months previously. Examination revealed a mass just within the anal sphincter, which

Fig. 17.—Plain roentgenogram of the pelvis in case 7, showing the distribution of radon seeds.

involved the sphincter and the anterior wall and both lateral walls of the rectum. The prostate was uninvolved. The microscopic appearance of the tumor is shown in figure 16. Dr. Curtis F. Burman implanted radon seeds in the tumor and gave a course of external roentgen irradiation (fig. 17). On February 10 Dr. Burnam applied radium directly to a small residual ulcer in which biopsy showed adenocarcinoma. Examination on May 25 revealed no evidence of recurrence.

CASE 8.—E. B., a white woman aged 70, consulted the clinic May 11, 1938. Pain in the rectum and constipation had been present for nine months; occasional blood in the stools, for two months. Examination revealed a lobulated mass 4 by 2 cm. in the posterior wall of the rectum, beginning at a point 5 cm. above the sphincter. The microscopic appearance of the tumor is shown in figure 18. My associate Dr. Eugene Covington treated this patient by direct application of radium and by external roentgen irradiation. At examination on June 13, 1938, approximately four weeks after the beginning of the irradiation therapy, no visible or palpable evidence of the nodular tumor remained. The appearance was that of a healing ulcer.

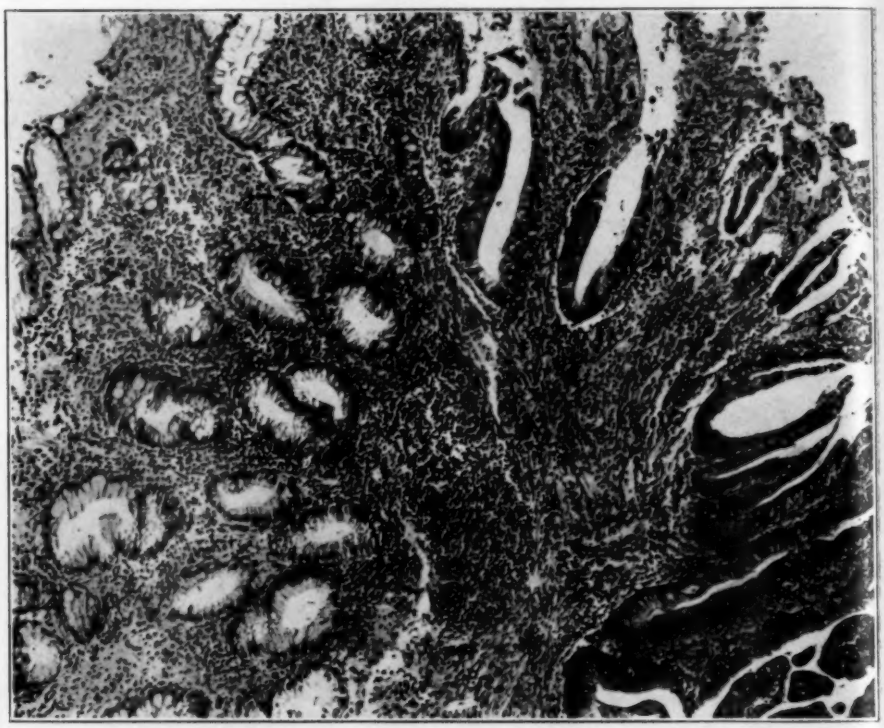

Fig. 18.—Low power photomicrograph of the tumor in case 8. At the left are normal glands; at the right, irregular glands with epithelial cells, showing hyperchromatic nuclei.

COMMENT

In spite of the fairly long duration of symptoms, the lesion in each of these cases presented the appearance of an operable carcinoma, and for this reason it is with some hesitation that I present such recent experiences with irradiation therapy. Because the immediate results in these 4 instances have been so striking I have great hope that further experience will justify a trial of irradiation therapy before operation in all cases of early operable carcinoma involving the lower part of the

rectum. My associates and I have not as yet had sufficient experience with irradiation therapy in cases of this kind to know exactly how the lesion should heal, but in none of the few cases observed by us has a stricture occurred, and the scars have been insignificant. The numerous statements in the literature that adenocarcinoma is not a radiosensitive tumor are apparently incorrect, because each of these 4 tumors proved to be an adenocarcinoma and yet completely disappeared under irradiation. Whatever the ultimate results may be, this fact cannot change.

REVIEW OF UROLOGIC SURGERY

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(Concluded from page 169)

URETHRA

Staphylococcic Infections.—Harkness and King[32] said that when staphylococci are found in the genital tract, cultures almost always show them to be of the albus type. Although Staph. aureus is rarely found, when it is the infecting organism the suppuration is more extensive and profuse and there is greater constitutional disturbance than occurs with Staphylococcus albus.

Pathogenic organisms are distinguished from contaminating organisms by the fact that they are present in large numbers, are the sole or predominant organism and give a profuse cultural growth.

Staph. albus is the commonest cause of primary nongonococcic urethritis. Such infection is often venereal. The incubation period is usually longer than in cases of gonorrhea except when the infection is superimposed on urethritis chemically induced by the use of strong solutions for prophylaxis.

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32. Harkness, A. H., and King, A. J.: Staphylococcal Infections of the Genital Tract in the Male, Brit. J. Urol. **10**:379-391 (Dec.) 1938.

There is a chronic type of staphylococcic urethritis in which the onset is insidious and the symptoms are mild; it may be overlooked even when there are pronounced granular changes in the urethral mucosa. Staphylococcic urethritis is relatively a more common cause of stricture than is gonococcic urethritis. Secondary staphylococcic urethritis following gonorrhea is common. Repeated cultures should be made of the secretions from the prostate gland, the seminal vesicles and Cowper's glands, although the focal lesions responsible for persistence are often in the anterior portion of the urethra.

Thus, urethritis following trauma from injections of strong solutions, careless or inefficient catheterization or urethral dilatation is usually abacterial at first but later shows Staph. albus on smear and on culture.

Urethral discharge caused by staphylococcic urethritis may be the only symptom of a serious lesion of the upper portion of the urinary tract, and this possibility should be borne in mind even if the patient states that there has been recent sexual intercourse. Obsessed with the possibility of venereal infection, the patient may overlook other symptoms of gradual onset and longer duration.

Treatment consists of through and through irrigations with any weak, warm and nonirritating antiseptic solution, followed by urethral dilations. The new chemotherapy profoundly modifies the course of the disease, and sulfanilamide combined with the irrigations is effective. When sulfanilamide fails, mandelic acid will frequently clear up the infection. Dilations followed by irrigations should be given once a week until urethroscopic examination shows them to be no longer necessary.

Prostatitis is the most common complication of primary staphylococcic urethritis, and urethral stricture may be a predisposing cause. The symptoms and clinical course of acute and chronic prostatitis, prostatic abscess, cowperitis, seminal vesiculitis and epididymitis caused by the staphylococcus vary little from those caused by other pyogenic organisms.

Infection of these structures with the staphylococcus follows gonorrhea, urethral instrumentation, descending renal infections or operation (prostatectomy), or the infection may be blood borne. Harkness mentioned 2 cases of prostatitis in which the condition followed a crop of boils. In neither case was there a history of infection of the urinary tract or venereal exposure.

In the acute stage of infection of these structures, sulfanilamide promptly relieves the symptoms. Acute prostatitis and cowperitis are likely to be more resistant to the drug, as is chronic staphylococcic prostatitis, although occasionally a dramatic cure may result.

Abraham [33] said that of the three types of staphylococci, Staph. aureus, Staph. albus and Staph. citreus, the first is pathogenic, the second is mildly so and the third is not a pathogen. Staph. aureus and Staph. albus are almost always present in the skin and in the sebaceous glands. To change them from saprophytes to parasites there must be trauma, local pressure or irritating discharge on a moist surface. Such conditions often involve the female genitalia.

In 100 women suffering from urethritis and endocervicitis, smears and cultures showed staphylococci in the cervix and urethra in 52, B. coli and diphtheroids in the cervix and urethra in 33 and staphylococci, diphtheroids and B. coli in the urethra in 10 and in the cervix in 8 of these 10 patients. All of them, in addition, were examined for Trichomonas vaginalis, but this was found in only 7 cases.

In children, infections of the genitalia exist as vulvovaginitis, because the glandular structures (Bartholin's gland, Skene's glands and the cervical glands) are not developed and because estrogen, which makes the vaginal epithelium resistant to infection, is absent.

In the vulvovaginitis of children, Staph. albus and diphtheroids are present in 25 to 50 per cent of cases. The discharge is thick, yellow and not offensive like that in infections with B. coli or Trichomonas. The lower third of the vagina is involved, and, although the external urinary meatus is inflamed, pain and increased frequency of urination are negligible. In adults these conditions are reversed.

Staphylococcic infections result in ductitis and bartholinitis, urethritis, skeneitis and endocervicitis. Forty per cent of infections of Bartholin's glands are staphylococcic or streptococcic. Staphylococcic urethritis is commonly secondary to gonorrhea; as a primary infection it is surprisingly rare. Vaginitis is rare, but staphylococcic endocervicitis is common.

In the treatment of these conditions, chemotherapeutic and bacteriologic agents are valuable. Manganese butyrate given intramuscularly in doses of 1 cc. of a 1 per cent solution every five days speeds up the disappearance of staphylococci from cervical and urethral smears. Abraham [33] said that in his hands the results of sulfanilamide therapy have not been impressive. Others have been more fortunate.

Formerly, because of the success of vaccine therapy, it was thought that the pathogenicity of staphylococci was due to endotoxins. Now it is known that they produce three toxins, alpha, beta and leukocidin, so that treatment by toxoids and antitoxin greatly augments ordinary vaccine therapy.

Antistaphylococcus serum is given intramuscularly in doses of 10 to 50 cc. daily after a minute dose has been given to see if the patient is allergic.

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33. Abraham, J. J.: Staphylococcic Infections in the Female Urethra and Genitalia, Brit. J. Urol. **10**:392-399 (Dec.) 1938.

Staphylococcus toxoid comes in two strengths, the weaker being one tenth as strong as the stronger. A preliminary injection of 0.5 cc. of the weaker toxoid is given subcutaneously, and if the reaction is not severe, 0.1 cc. of the stronger is given a week later. The dose can be increased weekly.

For staphylococcic vulvovaginitis of children, local treatment should consist in cleanliness, baths and mild germicidal irrigation. Over-treatment should be avoided.

For bartholinitis the glands should be opened, drained, packed and allowed to granulate from the bottom.

Ductitis and skeneitis are best managed by incising the ducts with a fine diathermy point.

For urethritis, injections twice weekly of glycerin, 10 per cent strong protein silver and glycerin or 1 per cent mercurochrome are valuable. In cases of chronic involvement the urethra should be dilated and 1 per cent silver nitrate instilled.

Acute endocervicitis is best treated by swabbing the cervical canal with glycerin, and using glycerin tampons and vaginal douches of 2 per cent lactic acid. Local applications in chronic endocervicitis should be escharotic to be of any value. For the same reason, diathermy, if employed, must be used as a cautery.

If there are lacerations, tracheloplasty is indicated.

Catheterization.—Emmett [34] discussed the minimal armamentarium and the points in anatomy and technic which may be of value in cases of difficult urethral catheterization. The article is intended primarily for general practitioners. Emmett mentioned the fixed and relatively inelastic roof of the urethra and emphasized the importance of having the instrument hug the anterior urethral wall during catheterization. Sterile technic, adequate lubrication and good catheters are necessary. Slowness and gentleness, especially at the sphincters, are essential. If the use of the soft rubber catheter is unsuccessful, Emmett advocated successively the use of a coude soft rubber catheter with a hollow olive tip, the woven coude or bicoude catheter, a soft rubber catheter with a hollow tip over a wire stilet or a filiform guide followed by a woven catheter. The value of morphine and a sitz bath as relaxing agents is stressed. If all these methods fail, anesthesia may relax the patient enough to allow passage of a catheter; this being unsuccessful, suprapubic drainage may be required. Proper use of the indwelling catheter is also considered.

34. Emmett, J. L.: Difficulties in Urethral Catheterization, Am. J. Surg. **40**: 349-356 (May) 1938.

PROSTRATE GLAND ?

Cancer.—Kahler [35] studied 195 carcinomas of the prostate gland, 72 of which were diagnosed clinically and the diagnosis confirmed at postmortem examination and the remaining 123 of which were diagnosed after the death of the patient. The clinical diagnosis was based on palpation alone in 23 cases, was supported by biopsy in 30 additional cases and in 19 cases was determined by palpation plus demonstration of distant metastatic lesions. In only 4 cases was a small tumor described clinically; 3 of the tumors were proved microscopically to involve the entire prostate gland and only 1 to be a small carcinoma (1 cm. in diameter). In this series the average age of the patients at the time of death was 68 years. The incidence of carcinoma of the prostate gland in men more than 50 years of age was 17.3 per cent. In only 3 cases in which the diagnosis was made clinically and 93 cases in which it was made at necropsy was the carcinoma confined to one lobe. In only 46 per cent of these was the posterior lobe the involved portion, as compared with 48 per cent in which the lateral lobes were involved and 6 per cent in which the anterior lobe was involved.

In only 53 per cent of the entire 195 cases was carcinoma recognized grossly at necropsy. Fifty-one per cent of the tumors were recognized because of increased consistency above the remainder or because of a yellowish, white or hemorrhagic tint with or without increased consistency. The yellowish tint, which is due to fat and urochrome pigment, was present in only 21 per cent of the tumors. On microscopic study, Kahler [35] found that all the tumors were adenocarcinomas except 3 per cent, which were squamous cell malignant growths. The grading of the adenocarcinomas was as follows: grade 1, 19 per cent; grade 2, 50 per cent; grade 3, 27.5 per cent, and grade 4, 3.5 per cent. The incidence of the lower grades was greater in the localized tumors and in the lower age groups. The most important microscopic criterion of prostatic cancer is involvement of the perineural lymphatics. This was observed in 91 per cent of the cases and in 100 per cent of those in which the tumor was graded 3 or 4. The localized tumors, even the smallest ones, showed the same high incidence of perineural involvement, an important argument against the feasibility of local removal.

Kahler [35] found that in 51 per cent of the clinically or grossly recognized tumors metastasis had occurred by the time death took place. The points of metastasis, in order of their frequency, were the lymph nodes, lungs, pelvic peritoneum and bone. Direct extension occurred, in order of frequency, to the bladder, ureters, seminal vesicles and rectum. The incidence of metastasis increased markedly as the grade of the

35. Kahler, J. E.: Carcinoma of the Prostate Gland, Proc. Staff Meet., Mayo Clin. **13**:589-592 (Sept. 14) 1938.

lesion increased, being 100 per cent in lesions of grade 4. The incidence of metastasis bore no relation to the size of the gland.

The association of atrophy and carcinoma was found to be incidental, as atrophy occurred in the same proportion of carcinomatous glands as of normal ones. Nodular hyperplasia also showed an incidental relation, as only 16.6 per cent of the localized tumors were found to have arisen from regions of nodular hyperplasia. Similarly, no relation existed between carcinoma and asymmetry, inflammation or calculi in the prostate gland.

Abscess.—Tomassini [36] stated that the most common factors that cause abscess of the prostate gland are inflammation of the bladder and especially of the urethra. Prostatic abscess usually follows acute prostatitis, which may be diffuse or localized. Although all authors agree that gonorrheal urethritis is the essential factor, the micro-organism most frequently present is a staphylococcus.

Diagnosis is made mostly by rectal exploration, the results of which are interpreted in connection with the history as regards past infection, the subjective disturbances and the temperature. Most frequently the abscess opens spontaneously into the urethra or rectum. The treatment of choice when the collection of pus is considerable and when the general condition is such as to permit it is perineal prostatotomy.

An abscess is occasionally found before puberty, when the gland has not acquired its special function. Two cases have been recorded in which the patients were young children, aged 4 years and 28 months respectively. The disposing factors are diabetes, gout, lymphatism, scrofula and arthritism. Chronic contusions, such as those caused by horseback riding, which provoke congestion of the veins of the small pelvis, may awaken a latent infection, most often in the urethra. In addition, the prostate gland feels the influence of all other causes of congestion, such as sedentary habits, hemorrhoids, constipation and proctitis, all of which favor stagnation of blood in the venous plexuses of the small pelvis.

The organ is enlarged, hyperemic and edematous. The microscopic picture varies according to whether the parenchyma or the interstitial tissue is chiefly involved. In a large number of cases the origin is in the glandular lacunas. If the collection of pus succeeds in opening spontaneously through the urethra or rectum, spontaneous cure occurs. In other cases cure is slow and may be delayed by purulent or urinary fistulas. In rare cases the prostate gland may be overwhelmed by a grave and rapid suppurative process, in which it and the surrounding tissues fall into a state of necrosis; in such cases the condition nearly always ends fatally.

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36. Tomassini, I.: Sull' ascesso della prostata, Arch. ital. di urol. **15**:292-299 (June) 1938.

When the abscess fails to open spontaneously or when the opening is insufficient and the tumefaction has reached a considerable size, it must be opened surgically. It may then be approached by the hypogastric, rectal or perineal route. The last is preferable, with a bi-ischiatic incision made horizontally between the rectum and the urethra. The abscess is reached, opened, carefully drained and treated with suitable medication.

The first symptoms are urinary and consist of frequency and urgency of voiding, soon followed by a sense of weight and tension in the prostate gland and rectum. The temperature is high, and chills sometimes occur. Rectal examination is extremely painful, a finding that distinguishes an abscess from a tumorous condition or hypertrophy. Fluctuation is a late finding. Frequently the sulcus cannot be found, and the gland feels like a single mass. The urine is only slightly cloudy until perforation into the urethra occurs, which causes the urine to become purulent. Such an opening may be spontaneous or may occur during catheterization or rectal exploration.

Emmett, Lovelace and Mann [37] carried out a series of intraprostatic injections of sclerosing solutions. The late result of injection of such solutions into the prostate glands of dogs was a definite reduction in the size of the lobe into which the solution had been injected. The reduction seemed to be associated with increase in the connective tissue stroma, possibly with some reduction in size and number of the prostatic acini. There was no untoward reaction, and symptoms referable to the urinary tract were not observed in any of the experiments. The article is a report of animal experimentation only, and its clinical application in any type of case is not suggested until further experimentation has been done. The work, however, does furnish a new field for thought, and the method will bear further investigation.

Hypertrophy.—According to Retlev-Abrahamsen and Aalkjaer,[38] many patients who have prostatic hypertrophy and who do not respond to treatment by drainage and forced fluids are considered genuinely uremic when, in fact, they are suffering from pseudouremia or nephrogenous acidosis.

This condition can be recognized by a determination of the value for plasma bicarbonate, which is normally 25 to 30 mg. per liter of blood. When this falls below 19 mg. per liter there are nausea, loss of appetite and dryness of the tongue; the patient cannot drink and is lethargic. When the value for bicarbonate is less than 14 mg. per liter, typical

37. Emmett, J. L.; Lovelace, W. R., II, and Mann, F. C.: Intraprostatic Injection of Sclerosing Solutions: An Experimental Study, J. Urol. **40**:624-628 (Nov.) 1938.

38. Retlev-Abrahamsen, H., and Aalkjaer, V.: The "Pseudouremia" of Patients with Prostatic Hypertrophy—The Nephrogenous Acidosis, Brit. J. Urol. **10**:231-236 (Sept.) 1938.

acidotic coma appears, with Kussmaul respiration. The clinical evidences of nephrogenous acidosis in cases of chronic prostatism are anhydremia, hypochloremia, fever, dyspepsia and senility.

Examination for and treatment of nephrogenous acidosis is indispensable to surgical treatment of the prostate. Treatment consists in the administration intravenously of 1.3 per cent (isotonic) sodium bicarbonate solution. The amount given is based on the blood value for bicarbonate and the body weight and is determined by use of the nomogram of Palmer and van Slyke. If according to this nomogram the patient is to be given 3 liters of a 1.3 per cent solution of sodium bicarbonate, 1 liter of the solution is given each day for three days and an analysis of the blood is made on the fourth day. In this way alkalosis and tetany are avoided.

Of 123 patients with prostatic hypertrophy treated by Retlev-Abrahamsen and Aalkjaer [38] since January 1936, one third were found to have nephrogenous acidosis. Appropriate treatment reduced the values for blood urea, corrected the anhydremia and permitted operation in a few days on patients who formerly had been considered hopelessly uremic.

Endocrine Therapy.—Walther and Willoughby [39] stated that prostatic hyperplasia can no longer be regarded as an independent entity; it is inseparably bound up with endocrine changes affecting the pituitary body and the testis.

In cases of early prostatism or in cases in which for some serious physical disability caused by any type of prostatic obstruction operation seems inadvisable, androgens should be given conscientious trial. The disadvantage of this mode of treatment is that, as with insulin therapy, one must continue a maintenance dose; therefore, contact with the patient for an indefinite period is necessary, and massage is usually indicated. Preparations of androsterone and testosterone propionate for oral and intramuscular use are available for such therapy.

Fifteen patients with benign prostatic hyperplasia have been treated with these substances by Walther and Willoughby [39] during the past two years, with clinical improvement of their symptoms.

Moore and McLellan [40] made a histologic study of the effect of androgen and estrogen on the prostate gland of the human being. They stated that the injection of 285 to 1,125 mg. of testosterone propionate in twelve to ninety-five days results in no significant restoration of the involuted prostate gland of presenility and causes no observable

39. Walther, H. W. E., and Willoughby, R. M.: Hormonal Treatment of Benign Prostatic Hyperplasia, Tr. Southeast. Br., Am. Urol. A., Nov. 5, 1937, pp. 63-72.

40. Moore, R. A., and McLellan, A. M.: A Histological Study of the Effect of the Sex Hormone on the Human Prostate, J. Urol. **40**:641-657 (Nov.) 1938.

alteration in the histologic appearance of the tissues formed in benign hypertrophy. The injection of 15,000 to 140,000 international units of estradiol benzoate in ten to thirty-one days produces conspicuous alteration in the urethral and ductal epithelium but little if any change in the tissues of benign hypertrophy.

Infarction.—Hubly and Thompson [41] found a single study (Abeshouse, 1933) of prostatic infarction in a review of the literature. The authors' report is based on a clinicopathologic study of 10 cases of prostatic infarction. Hubly and Thompson stated the opinion that function of the prostatic portion of the urethra is influenced by volumetric changes in the prostate gland and that changes of this type resulting from infarction may produce symptoms. The symptoms produced depend on the stage of infarction. During the early stage, when swelling is most pronounced, varying degrees of urinary obstruction, including acute urinary retention, may develop. In the later stage, cicatrization and contraction occur and the patient voids satisfactorily. This sequence of events, perhaps repeated on several occasions, may explain episodes of retention followed by more or less spontaneous relief of symptoms. The final stage of prostatic infarction may be manifested by regions of fibrosis, which are commonly found in the prostate gland. The 10 cases reported are considered in some detail. Prostatic tissue was obtained at necropsy in 6 of these cases and by transurethral resection in the remaining 4.

Trauma (such as that caused by prostatic massage), difficult urethral catheterization, prostatic resection and electrocoagulation may be factors in the production of infarction. Abeshouse has suggested that adenomatous hyperplasia, by distortion of the intraglandular blood supply, may cause infarction. Infection, circulatory stasis and arteriosclerosis may also be etiologic factors.

TESTICLE

Ectopy.—Jones and Lieberthal [42] stated that only 103 cases of perineal testicle have been reported. The exact mechanism of normal descent of the testis is not known, although the subject has been widely discussed and many elaborate hypotheses have been offered. It is known that before migration of the testes occurs, in early fetal life these glands occupy primarily a site in the lumbar region on either side of the vertebral column, in front of the psoas muscle and internal to the kidneys. At the beginning of the third month they begin to descend along the posterior abdominal wall, carrying with them their vascular pedicle and

41. Hubly, J. W., and Thompson, G. J.: Infarction of the Prostate: Its Clinical Significance, Proc. Staff Meet., Mayo Clin. **13**:401-403 (June 29) 1938.

42. Jones, A. E., and Lieberthal, F.: Perineal Testicle, J. Urol. **40**:658-665 (Nov.) 1938.

pushing ahead of them the parietal peritoneum, which is to constitute the processus vaginalis. As they gradually move downward they occupy successively positions in the abdominal, iliac and inguinal regions, until they finally reach their permanent bed within the scrotum just before or, occasionally, soon after birth.

A testis may become arrested at any of the first three stages; it is then known as an undescended testis. In addition to such arrests, however, there are found, in much rarer instances, deviations in migration which prevent the testicle from pursuing its normal course, deflecting it from its appointed path and landing it in some spot from which it cannot possibly reach the scrotum. When this occurs, the testis is known as ectopic. Unlike the testis that remains undescended, which is frequently the victim of some abnormality, the ectopic testis, according to most authorities, is usually normal and perfect in its development.

Ectopic testicles are of four varieties: (1) superficial, inguinal or interstitial; (2) penile; (3) perineal, and (4) crural or femoral. The perineal testis lies in practically the same position in all cases. It is always found between an imaginary line in front passing behind the root of the scrotum and a similar line passing in front of the anus; laterally, it is always outside the line of the raphe; it has never been known to lie behind the bi-ischiatic line, which passes anterior to the anal orifice. No adhesion of the testis to the tissues covering it has ever been observed. Frequently it can be slipped about with the greatest ease, sliding under the finger and often displaying sufficient mobility to pass, under pressure, into neighboring regions.

Treatment is surgical. The gland has gone into the wrong fascial pocket, and only surgical measures can restore it to its rightful bed. The cord in such a case is always long enough for this transplantation, and the operation is easily executed. If possible it should be carried out before the boy subjects the organ to trauma, which may easily occur with the testis in this perineal position. The operation should not, however, be carried out on children under the age of 3 years unless the condition is causing symptoms.

Jones and Lieberthal reported the case of a 17 year old boy. There was a mass about the size of a plum situated to the right of and close to the anal sphincter. While this mass was somewhat tender on palpation, there was no history of pain or discomfort, although the boy had taken part in various athletic games at school. The right side of the scrotal sac was empty and somewhat shrunken. At operation a normal testis was separated from its gubernaculum and placed in the scrotum.

Tumor.—McDonald [43] reported that approximately 142 cases of chorionepithelioma of the testes have been reviewed. He reported an

43. McDonald, S., Jr.: Observations on Chorionepithelioma Testis, with Record of a Case, Am. J. Cancer **34**:1-14 (Sept.) 1938.

additional case. The patient was a man 24 years of age. The right testis was three times the normal size. A diagnosis of teratoma having been made, the testis was removed. Histologic examination proved that the growth was a teratoma with chorionepitheliomatous elements. The qualitative Zondek-Aschheim reaction was positive eleven days after orchidectomy. Two months after operation the Zondek-Aschheim test showed 30,000 mouse units of gonadotropic substance per liter. The breasts had become moderately enlarged. The condition of the patient rapidly became worse, and he died three months after the operation. Necropsy showed multiple metastatic lesions, especially in the lungs and kidneys.

McDonald considered the histogenesis of the tumor and concluded that, although the tumor arises through malignant differentiation of a teratoma, endocrinologic observations support the belief that testicular chorionepithelioma is morphologically identical with uterine chorionepithelioma. He suggested that a quantitative Zondek-Aschheim test is essential in the investigation of testicular tumors and that correlation of the amount of gonadotropic substance present in the urine and the histologic characteristics of the growth may afford valuable information as to the nature and source of gonadotropic hormones.

Ormond [44] reviewed the symptoms and results in cases of torsion of the testicle and reported 12 new cases. He emphasized the importance of prompt recognition of this condition and stressed the following elements in the diagnosis: (1) the age of the patient [in this series 8 patients were less than 23 years of age, the youngest being 4 years old and 5 others being adolescents]; (2) the sudden onset; (3) the severity of the pain; (4) the absence of history or evidence of genitourinary infection; (5) the position of the affected testicle in the scrotum; (6) the position of the epididymis with reference to the testicle; (7) the tenderness of the testicle, and (8) Prehn's sign.

Finally, Ormond emphasized his conclusions regarding treatment. He stated that in an acute attack prompt operation offers the best chance of a healthy testicle; that although an attack is relieved by manual or spontaneous detorsion, operation should be done soon to prevent recurrence, and that if because of torsion a testis has become atrophic or has been removed, operation should be done on the remaining testis to prevent a like fate befalling it.

Hypertrophy.—Zide [45] studied 19 cases of unilateral testicular abnormality occurring after puberty. He wished to determine whether compensatory hypertrophy of the remaining testis actually occurred. In

44. Ormond, J. K.: Torsion of the Testicle, J. A. M. A. **111**:1910-1914 (Nov. 19) 1938.

45. Zide, H. A.: Does Compensatory Hypertrophy of the Adult Human Testis Occur? Proc. Staff Meet., Mayo Clin. **13**:268-269 (April 27) 1938.

17 of these cases unilateral testicular atrophy occurred after the orchitis of mumps; orchidectomy for unknown reasons had been performed on the remaining 2 patients. Measurements of the length and width of the unaffected testis of each patient were made with a caliper. As a control, the testes of 29 normal adult persons were measured. The testes of the control series averaged 3.8 cm. in length and 2.3 cm. in width. The uninvolved testes in the 19 cases of unilaterally atrophic or absent testes averaged 3.9 cm. in length and 2.5 cm. in width. The difference in size between the normal and the abnormal groups was found to be of no significance in this small series. The measurements of the largest testis in each group were within the normal limits for length but in both groups slightly exceeded normal width. It is concluded that the testis of the adult human being does not undergo any appreciable compensatory hypertrophy after atrophy or removal of its mate.

Cabot stated that many teachers have said that compensatory hypertrophy does occur after the loss of one testis in adult life, because it is known that this phenomenon occurs in the case of the kidney. He stated, however, that the cases are different, as it is important in the economy of the body that more renal tissue should be available for use under stress. Although this work is practically convincing to Cabot that the alleged occurrence of hypertrophy of the testis in the adult lacks a sound basis in observed fact, it does not answer the question of increased growth of a testis when its fellow is lost during childhood.

URINARY CALCULI

Pyrah and Fowweather [46] discussed the etiologic problems presented by calculi in recumbent patients.

The calculi at first are pasty masses deposited in the calices or in the pelvis; these "mud-stones," as they have been termed, may remain in this condition or may solidify into true calculi. The authors stated the opinion that the kidney itself suffers no permanent damage. The calculi vary to some extent in composition, which is largely dependent on the reaction of the urine, but all writers agree that phosphates of calcium are the principal substances found in these calculi.

Calculi in recumbent patients have been attributed to suppuration of bone. Urinary infection has been regarded by some authors as essential, but, although frequently found, such infection is by no means always present. The essential condition in the production of calculi on recumbency is the establishment in the urine within the kidney of a sufficient concentration of calcium ions together with the necessary reaction to allow the calcium to be precipitated as a salt in either the

46. Pyrah, L. N., and Fowweather, F. S.: Urinary Calculi Developing in Recumbent Patients, Brit. J. Surg. **26**:98-112 (July) 1938.

larger tubules or the calices of the kidney. The processes which create this condition are varied, and no doubt several come into play in any given case. Pyrah and Fowweather stated:

> The principal etiologic factors are: (1) release of calcium salts from the bones into the bloodstream as a consequence of: (a) generalized decalcification of the entire skeleton as a result of immobilization; (b) localized decalcification of bone near the site of the injury or infection. The calcium salts so released are excreted mainly by the urine; (2) increased concentration of calcium salts in the urine because of dehydration resulting from isolation and low intake of fluid, thus favouring precipitation; (3) stasis and inadequate renal drainage because of the enforced recumbent posture; (4) dietetic factors: (a) influence of diet and drugs on the reaction of the urine; (b) total amount of calcium taken by mouth; (c) deficiency in vitamin A and (d) possibly hypervitaminosis D; (5) infection: (a) in the urinary tract; (b) ascending urinary infection in the female; (c) in other parts of the body, that is, in bone and (d) constipation.

In most of the recorded cases, renal calculi have been found to have developed after many months or years of recumbency. There are theoretic reasons for supposing that their development actually commences early in recumbency, and it is probably true that if a stone does not develop early it will not develop at all. The first symptom is usually hematuria, and it occurs soon after the patient has been turned from the dorsal position to the ventral; the hematuria is often profuse. Renal colic occurs in a considerable number of cases, with or without bleeding.

The aim for the future must be prevention of the formation of calculus and of urinary infection. Decalcification of bone, dependent as it is on relative hyperemia, cannot be absolutely prevented, but it can be minimized by routine daily massage and active movements of the limbs not actually splinted. Large amounts of fluid must be supplied to the patient at regular intervals throughout the day. The fluid should either be neutral in reaction (such as water) or should be such that "ash" from the solid residue will be acid in reaction. In order that stasis of urine containing solid particles may not occur in the renal pelvis, the recumbent patient should be turned (either by tilting him to one side or by turning him into the prone position) at fairly frequent and regular intervals. In order to render the urine acid and thus maintain solution of the urinary calcium phosphate, a diet yielding an acid "ash" should be adopted as a routine. Vitamin A should be prescribed, although deficiency of vitamin A as an etiologic factor is not yet finally established. Constipation should be avoided by the use of aperients if necessary. Absolute cleanliness, particularly of the vulval and anal region of females in plaster casts, is vital if ascending infection is to be prevented. Prophylactic examination of the urine for erythrocytes should be made once each month, and their presence should be taken to indicate the necessity for active therapeutic measures against a possible calculus.

The object of treatment is to cause the renal calculi to go into solution or to disintegrate into tiny particles which will pass down the ureter and be excreted. If the urine is not infected, operation is contraindicated, as the stone can usually be made to disappear.

Calculi associated with gross infection of the urine require surgical intervention for their removal. Calculous pyonephrosis and calculous anuria form absolute indications for operation.

Chemical Composition of Urinary Calculi.—Jensen and Thygesen [47] examined 35 phosphatic urinary stones to determine their chemical structure. They carried out systematic qualitative analysis of all the stones. In a number of cases a quantitative analysis was also done and an acid-basic ash determination made. The following substances were found in the stones: (1) $MgNH_4PO_4.6H_2O$; (2) a colloidal phosphate of calcium with imperfect, apatite structure, containing 3 to 3½ equivalents of calcium per mol of phosphoric acid and some water, and (3) $Ca_3(PO_4)_2$.

The substances numbered (1) and (2) are the ordinary ingredients of phosphate stones and are more often found mixed than in a pure state. Two stones consisted entirely of $Ca_3(PO_4)_2$. Calcium carbonate, normal magnesium phosphate and the secondary calcium phosphates, which are often supposed to form phosphate stones, could not be found.

UROLOGIC DIAGNOSIS

Scholl [48] stated that various urologic conditions greatly resemble the clinical picture, and not uncommonly lead to the diagnosis, of chronic glomerulonephritis. The most common conditions are infection, obstruction and certain metabolic disturbances. The majority of these diseases are readily recognized if a complete urologic examination is carried out. The similarity of the various urologic conditions to chronic glomerulonephritis leads to a diagnosis of nephritis. The fear that urologic instrumentation may be followed by serious reactions frequently prevents a complete study that would indicate the true nature of the lesion. Such a fear is in most cases not warranted, as reactions rarely follow cystoscopic procedures with the rapid, accurate methods now employed and the use of recently devised, innocuous urographic materials. Carefully and gently carried out, such examinations cause no trouble even to a patient with severely damaged kidneys.

In all cases in which there is the slightest doubt as to the diagnosis the patient should be given the benefit of a complete urologic investigation. If this is thoroughly carried out, a small but definite group of

47. Jensen, A. T., and Thygesen, J. E.: Ueber die Phosphatkonkremente der Harnwege, Ztschr. f. Urol. **32**:659-666 (Oct.) 1938.

48. Scholl, A. J.: Urologic Conditions Simulating Chronic Glomerulonephritis, J. A. M. A. **111**:1421-1427 (Oct. 15) 1938.

patients in whose cases a diagnosis of chronic glomerulonephritis has been made will be found to have conditions that can be partially or completely relieved.

ANESTHESIA

Ferrin [49] studied a series of controlled clinical cases in which diothane hydrochloride had been used for urethral anesthesia both prior to and subsequent to urethral trauma. In 100 cases only 2 reactions were noted, and both of these were mild, subsiding without treatment. Neither diothane hydrochloride nor any other anesthetic agent should be used in the traumatized or normal urethra without careful supervision. In cases in which the use of an anesthetic agent locally is deemed desirable in spite of trauma, diothane hydrochloride appears to be the anesthetic of choice. The prolonged relief from pain following the use of diothane hydrochloride in the urethra is extremely valuable.

Alken [50] made a résumé of the experience of the large urologic clinic in Berlin (formerly headed by Prof. A. von Lichtenberg, now by Dr. Heckenbach) with peridural anesthesia in the last three years. In that time, nearly 2,500 urologic operations have been done with the use of peridural anesthesia. This anesthesia, which is connected with the names of Dogliotti, Guttierez and Kraas, is based on the following principle:

The anesthetic reaches the sensory nerves in the spinal canal outside the dura mater, causing anesthesia which has the completeness and advantages of spinal anesthesia without its drawbacks. The technic is simple. As an anesthetic agent, Alken [50] recommended a 2 per cent solution of pontocaine hydrochloride to which is added 15 drops of 1:1,000 epinephrine hydrochloride to each hundred cubic centimeters. The patient sits on a table with the head bent toward the knees as for spinal anesthesia. The needle is inserted in the midline. The mandrin is removed when the needle has gone 1 cm. deep, and a 10 cc. syringe filled with physiologic solution of sodium chloride is then fitted on the needle. The needle is made to advance slowly toward the spine while the operator is attempting to inject the saline solution. Nothing can be injected as long as the point of the needle is passing through the ligamentum flavum. As soon as the point of the needle comes into the peridural space, the pressure of the saline solution in the syringe falls to zero and the solution flows in freely, indicating that the point of the needle is in the peridural space and is pushing the dura mater ahead of it, reducing the chance of its perforation. Five cubic centimeters of the anesthetic solution is now slowly injected. This would be about the

49. Ferrin, J. W.: Use of Diothane Hydrochloride in Urologic Cases, J. Urol. **40**:666-671 (Nov.) 1938.

50. Aiken, C. E.: Peridural Anästhesie, Ztschr. f. Urol. **32**:649-659 (Oct.) 1938.

correct amount for spinal anesthesia. Ten minutes later another 10 cc. is injected, if the first injection was correct and has not produced spinal anesthesia. After another ten minutes, the remainder, 10 cc., is injected. The total dose, therefore, is 25 cc. To a small patient it is better to give only 20 cc.; to a large person, 35 to 40 cc. Thirty minutes after the first injection the anesthesia is complete. The anesthesia is best near the segments where the injection has been made. There is usually complete relaxation of the whole musculature. The site of injection for operations on the kidney and the upper portion of the ureter is in the interspace between the twelfth dorsal and the first lumbar vertebra; for the rest of the ureter, between the first and the second lumbar vertebra, and for the bladder, prostate gland and genitalia, between the second and the fourth lumbar vertebra. The anesthesia lasts for two and one-half to three hours. During the whole period of anesthesia the general condition of the patient remains satisfactory. In the first minutes there is usually a slight rise in blood pressure, but after twenty minutes the pressure falls 10 to 15 mm. of mercury below the original level. Alken [50] often adds a mild narcosis induced by an intravenous injection of 1 cc. eukodal-scopolamineephetonine (Merck).[50a] This anesthetic is used at the Berlin clinic in every procedure for which good anesthesia is needed, from painful cystoscopic procedures to the most difficult resections and plastic operations. Age, diseases of the heart and diseases of the circulatory system are not contraindications.

There is only one grave danger in peridural anesthesia, that is, perforation of the dura mater, which may pass unnoticed. If the perforation is clear and spinal fluid drops out of the needle, spinal or general anesthesia must be used. Inducing anesthesia by injection into a segment above or below should not be attempted, as the perforation in the dura does not close at once. In some old patients the dura mater is not elastic, so that the point of the needle may cause some trauma or may partially penetrate the dura. In this case the anesthetic agent may slowly penetrate into the spinal canal, causing late shock. As the amount of pontocaine which enters the canal is small, the shock quickly wears off after administration of a stimulant, and the operation can be performed. This penetration of the dura has happened 11 times in the last 1,000 cases.

In Alken's [50] opinion, peridural anesthesia is the ideal method for the urologic surgeon, because it combines simple technic, harmlessness, good effect and long duration and is suited for all urologic procedures, from cystoscopic examination to major operations.

50a. Eukodal is dihydroorycodeinone hydrochloride; ephetonine is an isomer of ephedrine.

URINARY EXTRAVASATION

Ravenel [51] discussed extravasation from the lower portion of the urinary tract. First he briefly reviewed the anatomic facts which directly influence its course. These are the fascial planes. The superficial perineal fascia consists of two layers, superficial and deep. The deep fascial layer forms a thin aponeurosis of considerable strength, continuous with the dartos of the scrotum, with the fascia of the penis and with Scarpa's fascia on the anterior surface of the abdomen; on either side it is firmly attached to the outer lip of the ischiopubic rami. Posteriorly this deep layer curves around the superficial transverse perineal muscles to blend with the base of the triangular ligament.

The triangular ligament, or urogenital diaphragm, is composed of two layers. The structure stretches almost horizontally across the pubic arch, so as to close in the front part of the outlet of the pelvis. The superficial layer is separated from the subpubic ligament by an oval opening for the transmission of the dorsal veins of the penis.

The posterior layer of the triangular ligament is really a continuation of the pelvic fascia across the pubic arch.

The fascia of Denonvilliers is an aponeurotic structure which is attached to the tip of the prostate gland and the triangular ligament and passes upward between the rectum and the prostate gland, bladder and seminal vesicles. It sends an investment to the seminal vesicles.

Buck's fascia forms a dense fibrous investment of the corpora cavernosa and corpus spongiosum in a figure-of-eight sheath which terminates anteriorly at the base of the glans penis and is delimited posteriorly by the triangular ligament, where it is in apposition with Colles' fascia. It is continuous above with the suspensory ligament of the penis. In Buck's original article he described the fascia as continuous with Colles' fascia, but Wesson, by means of injection experiments to simulate extravasation, found evidence that Colles' fascia, although rather adherent to Buck's fascia at the base of the penis, passes down separately from Buck's fascia and envelops the entire penis except the glans.

The clinical evidence of extravasation of urine from the lower portion of the tract varies with the site of rupture through which the urine escapes.

The site of rupture in the urethra is indicated by the course taken by the extravasating urine. Extravasation occurring from the pendulous portion of the urethra, when not rapid, may ulcerate through the fascial planes and form a fistula, or it may pass forward along the corpus spongiosum and involve the glans penis.

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51. Ravenel, J. J.: Extravasation from the Lower Urinary Tract, Tr. Southwest. Br., Am. Urol. A., November 1937, pp. 57-62.

When rupture of the urethra takes place in that part included between the attachments of the scrotum and the anterior layer of the triangular ligament, usually the bulbous portion, the course of infiltration is directed by the deeper layer of Colles' fascia. This is the common site of rupture of the urethra when infection and obstruction are the etiologic factors. The extravasating urine here being limited by the deeper layer of Colles' fascia, it fills first the perineum just posterior to the scrotum; then it proceeds up over the symphysis to the abdominal wall, infiltrating beneath Scarpa's fascia.

Because of the close fusion between Colles' fascia and Buck's fascia at the base of the penis laterally and inferiorly, it is usual that the extravasation does not at first involve the penis. After reaching the abdominal wall, however, it descends to and involves the penis.

Rupture of the membranous urethra with extravasation between the layers of the triangular ligament is unusual and is rather difficult to diagnose in the early stages. Generally it is not until one layer of the triangular ligament gives way or until the extravasating urine reaches and emerges through the subpubic hiatus in the anterior layer that definite symptoms appear. When this occurs, the course is the same as when the bulbous urethra ruptures. Should the posterior layer give way, the urine may either follow the course of the rectum and appear at the anal perineum or pass up and invade the prevesical space.

Rupture of the urethra posterior to the deep layer of the triangular ligament is generally the result of trauma, such as is often seen in cases of fracture of the pelvis.

The prognosis depends as much on an early diagnosis as on proper surgical treatment.

Ravenel observed a series of 57 cases with a recovery rate of 65 per cent. The penis alone was involved in 7 cases; the perineum alone in 1; the perineum and scrotum in 5, the scrotum alone in 3 and the scrotum and penis in 16. Extensive infiltration of the perineum, scrotum, abdominal wall and penis was present in 23. There were 2 cases of traumatic rupture of the membranous urethra with extravasation between the layers of the triangular ligament.

Early operation is imperative to divert the stream of urine from the rupture and to provide free incision and drainage of the infiltrated portions. In 47 of the cases external urethrotomy was performed; in 3, suprapubic cystotomy and in 6, drainage by catheter. In 1 case operation was not done, the patient having died within an hour after admission to the hospital.

Ravenel based his preference for the perineal approach on the following facts: (1) there is dependent drainage; (2) there is less shock,

and (3) there is avoidance of contaminating the prevesical space with the gas-producing anaerobic organisms so often found.

DRUG AND FEVER THERAPY

Elkins and Krusen [52] mentioned the decline in the use of fever therapy for gonorrhea since the advent of sulfanilamide. A small group of patients who do not show a satisfactory response to sulfanilamide, however, are still treated by artificial fever. The authors cited 2 cases of gonorrheal infection in which artificial fever therapy failed to effect a cure but treatment with sulfanilamide was successful. They also reported 10 cases in which fever therapy was employed, in all of which an adequate amount of sulfanilamide had been given previously without producing the desired result. All but 1 of the patients were cured after the fever treatments.

Elkins and Krusen stated that recently sulfanilamide combined with artificial fever therapy has been employed. They considered Ballenger's plan of combined treatment, giving 80 grains (5.17 Gm.) of sulfanilamide for two days before artificial fever therapy was instituted. The temperature of the body was raised to 103 or 104 F. for a period of three to four hours. Ballenger reported good results from the use of this combination of methods, although Kendell stated the opinion that the combined treatment has little more effect than fever therapy alone.

Elkins and Krusen reported only 2 clinical remissions following the use of a combination of sulfanilamide therapy and five hour sessions of artificial fever at 106.7 F. in a series of 10 cases. They did not feel that from this series they could draw definite conclusions; however, they concluded that fever therapy, with or without sulfanilamide therapy, will continue to be an important adjunct in the treatment of certain resistant types of gonorrhea.

Cook [53] stated that, although the gonorrheal patients seen at the Mayo Clinic are likely to have the more "difficult" types of infection, cure of more than 90 per cent of these patients was obtained by the use of sulfanilamide alone or in combination with local treatment. If, after trial of the drug for ten days, there has not been definite improvement, fever therapy must be considered and will prove of great benefit. Time alone will tell whether treatment with sulfanilamide and artificial fever has any advantage over fever therapy alone.

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52. Elkins, E. C., and Krusen, F. H.: Fever Therapy in Resistant Gonorrhea, with Especial Reference to Its Relationship to Sulfanilamide Therapy of Gonorrhea, Proc. Staff Meet., Mayo Clin. **13**:299-303 (May 11) 1938.

53. Cook, E. N., in discussion on Elkins and Krusen.[52]

Emmett [54] called attention to the fact that the intravenous administration of mercurochrome has given disappointing results in the treatment of infections of the blood stream; however, small doses of this drug have been found useful in the management of acute pyelonephritis. Emmett mentioned the work of Braasch and Bumpus in 1926, in which reactions occurred in a large percentage of cases in which even a relatively small dose of mercurochrome was given intravenously. In most cases of acute pyelonephritis observed by Braasch and Bumpus the temperature returned to normal after the drug was given. In 28 of 69 cases in which the temperature was elevated on administration, the temperature returned to and remained at normal. There was no effect other than this antipyretic action. Two deaths were reported.

The constant need for some therapeutic agent to terminate a severe septic temperature in cases of protracted acute pyelonephritis gradually led to a reconsideration of the drug. Because the amount of mercury present in the blood stream during the administration of mercurochrome was shown to be exceedingly small, the effect has been felt to be nonspecific. If this were true, a smaller dose was thought to be of probable value. Accordingly, a small dose of 5 to 10 cc. of a 1 per cent solution has been used to terminate the high temperature associated with acute pyelonephritis.

Emmett stated that since 1933 mercurochrome has been used thus in about 125 instances, with only 1 known (moderate) reaction. The administration is made usually by diluting it with 500 cc. of physiologic solution of sodium chloride. The response is usually prompt and dramatic.

Emmett analyzed the results in 34 cases in which forty intravenous injections of mercurochrome were given. Valuable conclusions were drawn in spite of the evident inaccuracies. In some cases of high postoperative temperature, when the cause of the fever was in doubt, mercurochrome was administered. If crises of the fever occurred, it was felt that the diagnosis of acute renal infection could be made. The treatment was given in 22 cases of acute pyelonephritis, in 10 of which the condition was a postoperative occurrence. In 20 of these cases the fever was decreased, in 18 by crises. In 8 of these the fever recurred. In 5 of these 8, a second injection of mercurochrome was given and resulted in diminution of fever in 4 instances.

Emmett concluded that acute pyelonephritis is one condition in which the intravenous administration of mercurochrome may be expected to be effective. Although its only action is antipyretic, it may prove to be a life saving measure in the occasional case in which a septic fever threatens the life of a patient. In a large percentage of cases the acute

54. Emmett, J. L.: The Antipyretic Action of Intravenous Administration of Mercurochrome in Acute Pyelonephritis, J. Urol. **40**:312-318 (Aug.) 1938.

phase of the disease is terminated; however, the urinary infection must be eradicated subsequently by other chemotherapeutic means.

Strauss [55] called attention to the beneficial effects of the combination of calcium and bromides on inflammation of the uropoietic systems of nervous patients. He used the preparation calcibronat "Sandoz."

VAGINITIS

Schauffler, Kanzler, and Schauffler,[56] in a review of their cases of vaginitis in infants, stated that their experience with distention with silver nitrate ointment in 99 cases, vaginal application of amniotin (an estrogen) in 31 cases, insertion of pyridium suppositories in 19 cases and various other methods in smaller series leads to the conclusion that the use of amniotin by vaginal application is the most satisfactory method of management they have used.

Their study includes 261 cases in which sulfanilamide was administered orally. The results and opinions indicated that the method is unsatisfactory as used at present. The reason may be that administration of the drug is inadequate or inconstant. The desired low p_H of the vaginal secretions may be important in relation to the ineffectiveness of sulfanilamide. Meticulous care during treatment requires hospitalization, a disadvantage. The method as used thus far apparently does not compare favorably with other available methods.

Evidence from a rather painstaking study indicates that the endocervix is seldom an important factor in relation to vaginitis and is practically never such a factor when the patient is a young child.

HEMOSTASIS

Rannert [57] studied the hemostatic effect of vitamin P in different urologic conditions. She found that after intravenous injection of 55 mg., or after oral administration of 100 mg., of vitamin P, bleeding from any source in the urologic tract could be diminished or stopped. Other examinations revealed that the hemostatic effect was prompt when it was possible to raise the calcium content of the blood by 1.5 mg. per hundred cubic centimeters. If that rise did not take place, further administration of vitamin P was necessary until the higher level of calcium was obtained.

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55. Strauss, H.: Ueber kombinierte Brom- und Calciumbehandlung in der urologischen Balneologie, Ztschr. f. Urol. **32**:689-694 (Oct.) 1938.

56. Schauffler, G. C.; Kanzler, R., and Schauffler, C.: Management of Two Hundred and Fifty-Six Cases of Infection of the Immature Vagina, J. A. M. A. **112**:411-416 (Feb. 4) 1939.

57. Rannert, M.: Die blutstillende Wirkung des Citrins (P-Vitamin), Ztschr. f. Urol. **32**:630-633 (Sept.) 1938.